Risk factors for cardiovascular disease among undergraduate students in Edinburgh, Scotland

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ABSTRACT

An exploratory descriptive survey with a correlational design examined the risk factors for cardiovascular disease among a convenience sample of university students at Heriot-Watt University, Edinburgh. The first survey, with 156 students, identified several risk factors, including obesity, low of physical inactivity, consumption of alcohol and soft drinks with extrinsic sugars, not eating fruit and vegetables, and smoking tobacco. The second survey, with 40 students, provided more detailed information on dietary factors analysed by cluster and principal component analysis. The students were classified into four groups. One group (7.5% of the students) exhibited the greatest level of risk factors, including the highest BMI, body fat, waist to hip ratio, blood pressure, LDL-cholesterol, fasting glucose, the lowest HDL-cholesterol, the highest mean intake of alcohol, chloride saturated fatty acids and total sugars and intake of dietary fibre. The relationships between the risk factors were revealed by a structural equation model with a moderate effect size (R^2 = 48.9%). This model indicated that the BMI, body fat, and waist to hip ratio were higher if the students were smokers and consumed large amounts of alcohol, saturated fatty acids, salt, and sugars. The anthropometric measurements were also higher if the students had high levels of blood pressure, blood glucose, and LDL-cholesterol. The anthropometric measures were lower if the students consumed high amounts of dietary fibre, consumed at least five servings of fruit and vegetables every day, and had a high level of physical activity. More research and education including a health education programme based on a plan-do-study-act cycle is recommended to improve student awareness of their exposure to multiple risk factors for cardiovascular disease, and to determine how these risk factors can be alleviated in the future.

ACKNOWLEDGEMENTS

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DECLARATION

I hereby declare that I, Nada Alamri, am the author of this thesis. All the work described in this thesis is my own except where stated in the text. Results presented in this work have not been used in any previous application for a higher degree. All sources of information have been consulted by myself and are acknowledged by means of references.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

β	Path coefficient
BMI	Body mass index
CHD	Coronary heart disease
CVD	Cardio-vascular disease
3-D	Three-dimensional
DRI	Dietary reference intake
DRV	Dietary reference value
ELISA	Enzyme-Linked-Immune-Sorbent-Assay
HDL	High density lipoprotein
LDL	Low density lipoprotein
Max	Maximum
Min	Minimum
mm Hg	Millimetres of Mercury
n	Number of participants in a group
OGTT	Oral Glucose Tolerance Test
PC1	First principal component
PC2	Second principal component
PC3	Third principal component
PCA	Principal component analysis
PDSA	Plan-Do-Study-Act
PLS	Partial least squares
R^2	R squared, measuring the proportion of variance explained
SD	Standard deviation
SEM	Structural equation modelling
UK	United Kingdom
USA	United States of America
VLDL	Very low-density lipoprotein
WHO	World Health Organization

Chapter One

Introduction

1.1 Cardiovascular disease

This study focused on the risk factors for cardiovascular disease (CVD) among undergraduate students at Heriot-Watt University, Edinburgh, Scotland. A risk factor is defined as "any attribute, characteristic or exposure of an individual that increases the likelihood of developing a disease or injury" (WHO, 2015a). CVD is the general name for a family of diseases concerning disorders of the heart and blood vessels (Mendis et al. 2011). The most prevalent types of CVD are (a) coronary heart diseases (CHD) including angina (caused by an inadequate blood supply to the heart); and myocardial infarction (commonly known as a heart attack, when blood flow stops to part of the heart, causing damage to the heart muscle); (b) ischemic and haemorrhagic stroke (when a blood vessel that feeds the brain gets blocked or bursts) and (c) peripheral artery disease (disorders of the blood vessels that supply blood to the arms and legs). Other types of CVD include (a) congestive heart failure (when the heart does not pump blood as well as it should); (b) arrhythmia (an abnormal rhythm of the heart, including bradycardia, when the heart rate is less than 60 beats per minute, tachycardia, when the heart rate exceeds 100 beats per minute, and atrial fibrillation, characterized by abnormal irregular beating); (c) heart valve dysfunction, including stenosis (when the heart valves do not open enough to allow the blood to flow through); regurgitation (when the valves do not close properly and allow blood to leak through); and mitral valve prolapse (when the valves bulge back into the upper chamber of the heart); (d) cardiomyopathy (when the heart muscle becomes overly thick or stiffens); and finally (e) heart infections (e.g., pericarditis, myocarditis, and endocarditis).

The underlying mechanisms vary depending on the type of CVD. The most common types of CVD (CHD, stroke, and peripheral artery disease) are related to the

process of atherosclerosis when the walls of the arteries thicken, through the accumulation of white blood cells, and the proliferation of smooth muscle cells creating a plaque. Atherosclerosis has been associated with many risk factors for CVD (McGill et al. 2008). Some of these risk factors, cannot be changed, such as family history, ethnicity, gender, and age. Other risk factors, of specific interest to the current study, are modifiable, because they can be treated, or eliminated by changes in lifestyle. These factors include obesity, high blood pressure (hypertension), high cholesterol, diabetes, unhealthy diet; low levels of physical inactivity, excessive alcohol consumption; and smoking tobacco, particularly cigarettes (Hildreth et al. 2009). According to the World Health Organization (2015b) not smoking, engaging in physical activity for at least 150 minutes per week, eating at least five servings of fruit and vegetables a day, and limiting salt intake to less than one teaspoon a day will help to prevent CVD. Because CVD is preventable, by means of healthy eating, exercise, avoidance of tobacco smoke, limiting alcohol consumption, and treating high blood pressure and diabetes, it is important to identify the risk factors for CVD early in life, in order to reduce the risk of CVD in middle to old age (McGill et al. 2008; Paul et al. 2015).

1.2 Global mortality from cardiovascular disease

Figure 1.1 illustrates that the total number of global deaths due to CVD in 2008 was 17.327 million, with the highest rates in Europe (4.584 million) and the Western Pacific (4.735 million) (World Heart Foundation, 2015). Cardiovascular diseases, particularly CHD and stroke, are the most frequent causes of mortality in all regions of the world, apart from Africa (Mendis et al. 2011). In 2013 almost 30% of all deaths worldwide were caused by CVD, increasing from 12.3 million per year in 1990 to 17.3 million per year in 2013 (Global Burden of Diseases Study, 2013).

According to the World Health Organization (2015) more than 23 million people will die annually from CVD by the year 2030.

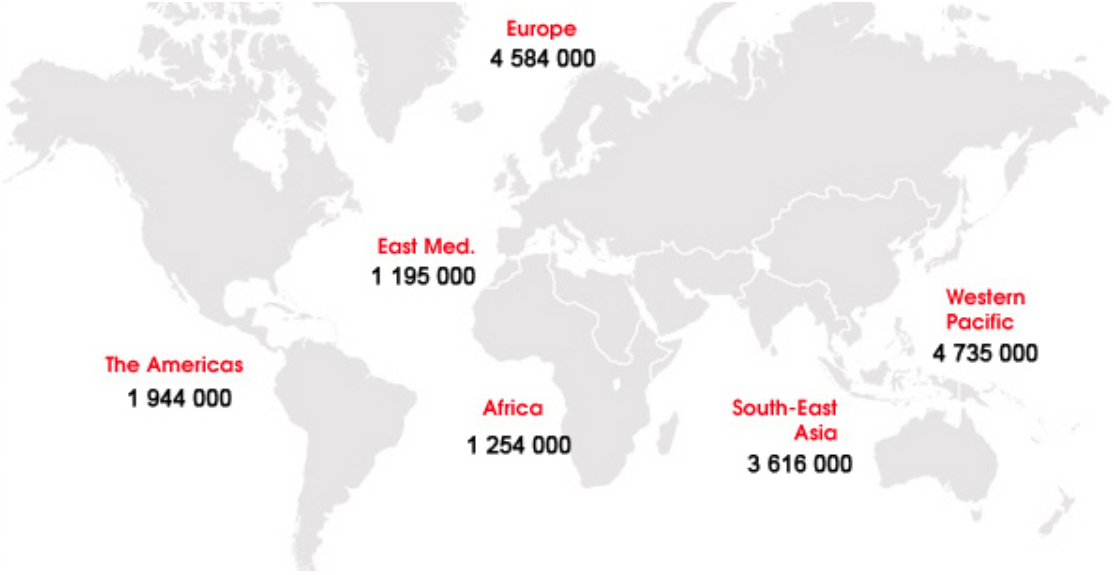

Figure1.1. Death rates due to CVD in the world in 2008
(http://www.world-heart-federation.org/cardiovascular-health/global-facts-map/)

1.3 Mortality and prevalence of cardiovascular disease in UK

Age-specific death rates from CVD in the UK were estimated by the British Heart Foundation (2014). Figure 1.2 provides a map of the age-standardized death rates from CVD per 100,000 in men and women of all ages by UK region in 2010 to 2012. Inequalities in mortality due to CVD occur across different regions of the UK. The death rates from CVD in the North of England, Midlands and Wales (279 to 321 per 100,000) were higher than in the South East and South West of England (269 to 279 per 100,000). The highest death rates were in Scotland (322 to 437 per 100,000).

The prevalence of CVD in the UK was analysed by Bhatnagar et al. (2015). In 2012 to 2013, about 2.3 million people in the UK suffered from CHD, about 1.2 million from stroke, about 1 million from atrial fibrillation and about half a million from congestive heart failure. Figure 1.3 displays the variations in the prevalence of

CVD in the UK from 1988 to 2011, in men and women, stratified by age. The trends indicate an increase in CVD prevalence for both men and women aged over 75 and for men aged 65–74. CVD prevalence for men aged 16–64 remained relatively constant, but for women of age 45–64 there was a small decrease from 10.8% to 8.4%

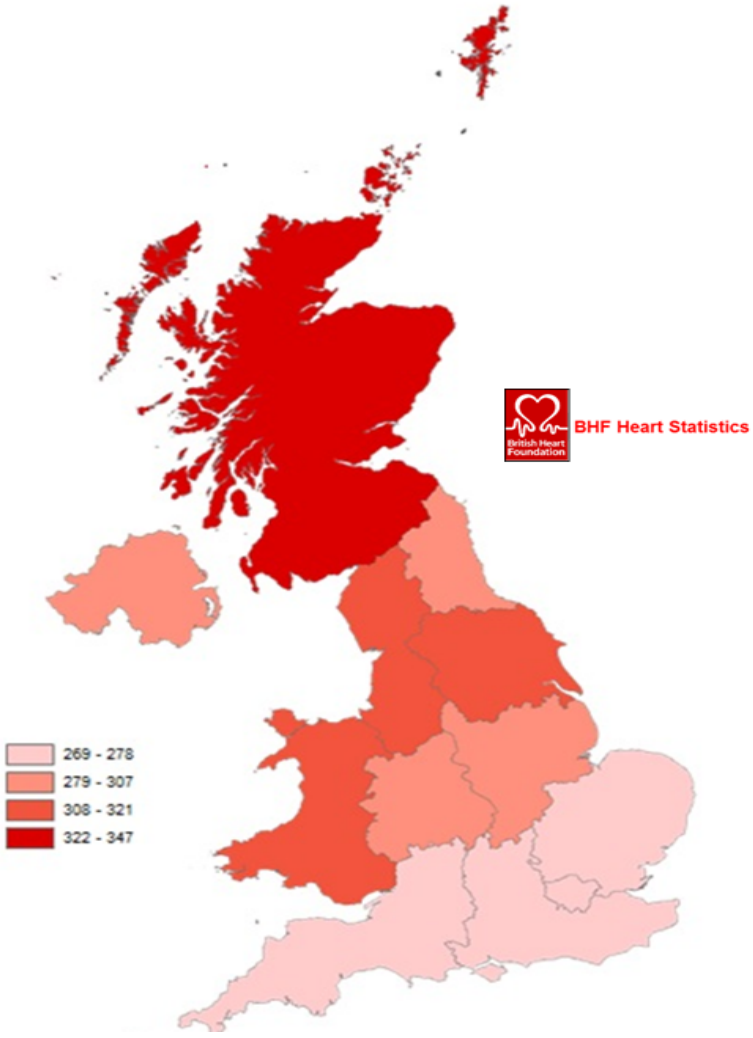

Figure 1.2 Age-standardized death rates from CVD / 100,000 in men and women of all ages by UK region in 2010-2012

(https://www.bhf.org.uk/~/media/files/publications/research/bhf_cvd-statistics-2014_web_2.pdf)

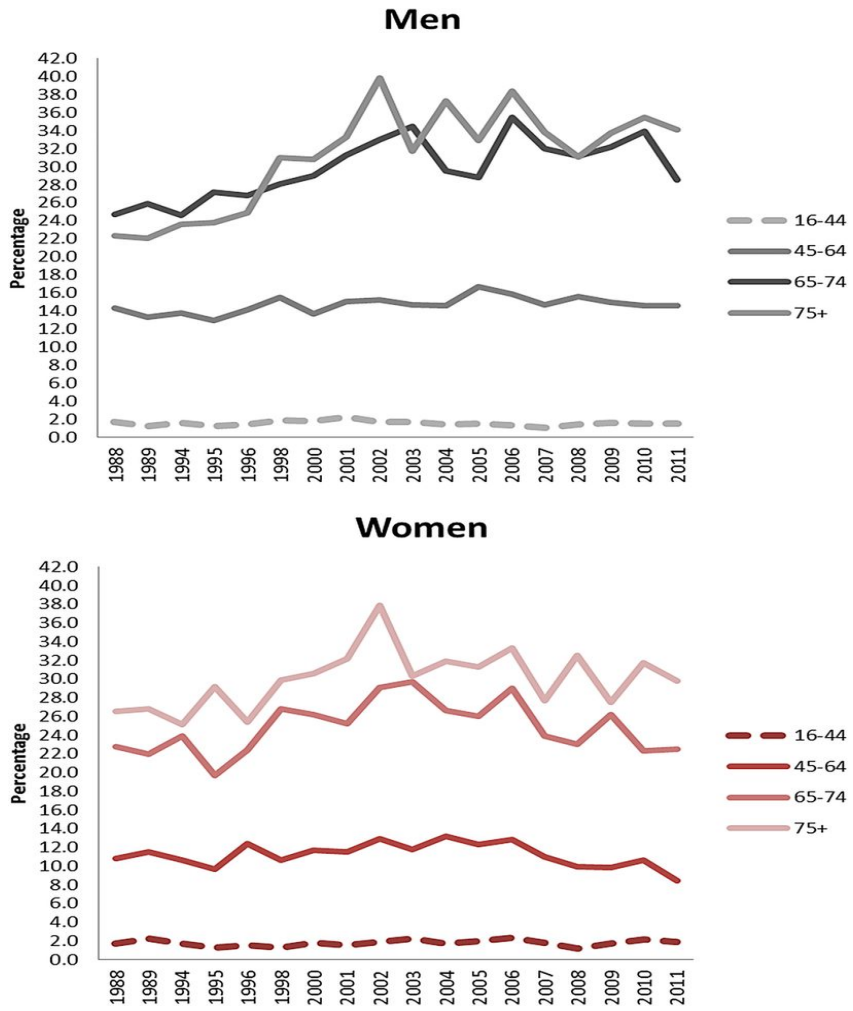

Figure 1.3 Trends in CVD prevalence in men and women in UK by age
(Bhatnagar et al. 2015)

CVD remains the highest cause of mortality among women in the UK. England has the lowest prevalence of CVD (3.4% for CHD, and 1.7% for stroke). The highest prevalence of CVD is in Scotland, at 4.3% for CHD, 2.1% for stroke, and 0.9% for peripheral arterial disease. The Scottish Health Survey (2011, 2014) confirmed that Scotland has the highest prevalence of CVD in the UK (322 to 347 per 100,000). Scotland has the highest CHD prevalence rate in Europe, and CHD is the most important cause of premature mortality in Scotland. Furthermore, the highest proportion of hospital inpatient episodes for all CVD recorded in the UK have been recorded in Scotland (12.4% of men and 8.6% of women). By city and gender, the

highest age-standardized death rates from CVD per 100,000 in the UK were in Glasgow, Scotland (515.9 male, 315.1 female) and Aberdeen, Scotland (413.0 male, and 302.4, female). In Edinburgh, Scotland, where the current study was undertaken, the death rates from CVD fell not far short behind Glasgow and Aberdeen (387.8 male and 268.3 female).

The mortality rate in most regions of Scotland is higher compared to England and Wales. Excess mortality in Scotland is particularly prevalent among young adults (16–44 years) and is observed across all occupational social classes with the greatest excess in unskilled workers (McCartney et al., 2015). Although Scotland has a higher mortality rate than England and Wales, this is only partially explained by differences in socio-economic deprivation. Scottish born immigrants to other countries have been shown to have a higher mortality rate than those born in England and Wales, suggesting that country of birth is more important than country of residence, and that early life factors or lifestyle choices may be important to explain the excessive mortality in Scotland (Popham et al. 2010). Significantly, there is a significant excess of CVD risk among South Asian immigrants in Scotland, compared to that reported in England and Wales (Fischbacher et al. 2007), suggesting that environmental or social factors in Scotland may play a role.

1.4 Expenditure on CVD in UK

Bhatnagar et al. (2015) reported that the prevalence of CVD in the UK is a considerable burden, both in terms of the health of the population and the costs for healthcare. The National Health Service in England spent about £6.8 billion on CVD in 2012 to 2013. The increased survival rates of patients with CVD, due to improvements in primary treatment, which have developed in the last twenty years, have resulted in a higher frequency of prescriptions for secondary prevention. Drugs

for patients with high risk of CVD are currently the most prescribed medicines for men, and the second most prescribed for women in the UK. The burden of CVD in Scotland is very substantial. The estimated annual cost to the National Health Service in Scotland exceeds £3 billion per year (Hotchkiss et al. 2014). Consequently, any preventative measures implemented to reduce the prevalence of CVD in Scotland should have important social, economic, and public health consequences. The control of risk factors for CVD is a critical preventative measure

1.5 Clinical risk factors for cardiovascular disease

Four of the most important clinical CVD risk factors include the following: First, a high level of LDL-cholesterol in blood serum. LDL-cholesterol is a lipoprotein that can lead to a build-up of plaque in the arteries. Atherosclerosis is the underlying cause of heart attack and stroke. Hyperlipidemia is the name given to the condition of excessively high LDL-cholesterol in blood serum (Linton, 2015). Second, hypertension or high blood pressure, which is a multifactorial disorder defined as a combination of excessively high levels of systolic and diastolic blood pressure, with mechanistic connections to other disorders including CVD and diabetes (Grundy et al. 1999). Third, diabetes, which is a family of diseases identified by high blood sugar (glucose) because the body cannot produce sufficient insulin or is unable to use insulin effectively (Diabetes UK, 2016). Fourth, obesity, which is generally defined as a body mass index (BMI) greater than or equal to 30 kg/m^2. The correlation between obesity and high levels of LDL-cholesterol was identified as a major risk factor for CVD by Wilson et al. (1997) and this correlation was confirmed in subsequent studies Canoy et al. 2013). The next four sections provide more detailed information on cholesterol, hypertension, diabetes, and obesity as risk factors for CVD.

1.5.1 Cholesterol

Cholesterol is a steroid lipid that occurs in all cells of the body. Cholesterol is essential to synthesize hormones, vitamin D, and bile for digestion, and is a major component of the plasma membrane. Most cholesterol is synthesized in the liver, but some cholesterol is derived from the diet, including egg yolks, fatty meats, and cheese. Cholesterol travels through the bloodstream in lipoproteins, consisting of fat (lipid) on the inside, and proteins on the outside. There are two types of lipoproteins carrying cholesterol, termed low-density lipoprotein (LDL-cholesterol) and high-density lipoprotein (HDL-cholesterol). LDL-cholesterol is commonly called "bad" cholesterol, because it leads to a build-up of cholesterol in the arteries. HDL-cholesterol is sometimes called "good" cholesterol, because it carries cholesterol to the liver, where it is removed from the body. The terms "good" and "bad" cholesterol, however, are misnomers, because LDL-cholesterol and HDL-cholesterol are not different types of cholesterol, but are different types of lipoproteins (American Heart Association, 2014a).

Hyperlipidemia is the name given to the condition of excessively high LDL-cholesterol and triglyceride levels in blood serum. As many as two thirds of the UK adult population may have hyperlipidemia (JBS3, 2016). Hyperlipidemia by itself does not cause any symptoms. High LDL-cholesterol is, however, associated with CVD because it leads to the build-up of plaque inside the arteries, a condition known as arteriosclerosis, or hardening of the arteries. Plaque is made of cholesterol, fatty substances, cellular waste products, calcium and fibrin, a blood clotting material (American Heart Association, 2014, b). Over time, the plaque hardens and narrows

the arteries, limiting the flow of oxygen-rich blood to the heart and other organs**.** An area of plaque may rupture causing the formation of a local clotting of blood known as a thrombus or thrombosis. If the clot becomes large enough, it can block the flow of blood through an artery **(**Furie & Furie, 2008). If the oxygen supply to the heart muscle is reduced in the coronary artery, a heart attack can occur. If the oxygen supply to the brain is cut off, then a stroke can occur. If the oxygen supply to the extremities is reduced, then gangrene can occur

Longitudinal research on cholesterol as a risk factor for CVD was provided by the Framingham Heart Study, which began in the 1950s in the USA, and continued for over 50 years (Wilson, 2013). The Framingham study focused on lipid measurements in blood plasma, their associations with disease risk factors, and specifically how they were related to the risk of CVD. In the initial Framingham Heart Study, only simple laboratory measurements, such as total cholesterol were analysed. The presence of total blood cholesterol >260 mg/dl was proposed by Kannel et al. (1971) as one of the "factors of risk" for CVD. Over time the methodologies progressed to lipoprotein analyses for the estimation of CVD risk. During the 1970's it was firmly established that a high risk of CVD occurred if the LDL-cholesterol level was higher than 50 to 70 mg/dL. A high level of HDL-cholesterol (≥ 60 mg/dL) indicated a low risk for CVD, because it protected against disease, by scavenging and removing LDL (Gordon et al. 1977). Research in the 1980s demonstrated that the risk of CVD could be predicted with reasonably accuracy using LDL-cholesterol and HDL-cholesterol measurements (Anderson et al. 1987). Research in the 1990s incorporated lipoprotein data into multivariate predictive statistical models along with other factors, to predict the risks of developing CVD outcomes.

Equations to predict the risk of CVD and death from CVD demonstrated the potential importance of controlling multiple factors (e.g., obesity, blood pressure, total cholesterol, LDL-cholesterol, HDL-cholesterol, smoking, glucose intolerance, and left ventricular hypertrophy) as opposed to focusing on one single risk factor. (Anderson et al. 1991; Wilson et al. 1997; 1998). The criteria currently recommended by the Executive summary of the third report of the National Cholesterol Education Programme (2001) classify LDL-cholesterol using a point scale: Optimal (< 100 mg/dL); Near/Above Optimal (100-129 mg/dL); Borderline High (130-159 mg/dL); High (160-189 mg/dL); and Very High (> 190 mg/dL). HDL levels are classified into Optimal (> 60 mg/dL); Near Optimal (40-59 mg/l) and Sub-optimal (< 40 mg/dL). The relative risk of CVD is also indicated by the ratio of LDL to HDL, and the ratio of HDL to total cholesterol.

Further research in the 21st century, concluded that alternative markers (e.g., apolipoprotein A and D) did not provide greater discrimination in estimation for risk of CVD than HDL and LDL (Ingelsson et al. 2007). The most recent research has focused more closely on the genetic risk factors (e.g., mutations, genomes, polymorphisms and alleles) associated with cholesterol metabolism that may increase the risk of CVD (Kathiresan et al. 2008; Whitfield, 2014; Mi et al. 2011).

Despite many years of research, the utility of various lipoprotein cholesterol measurements and how they may be used to predict CVD is still a subject of debate. For example, the total cholesterol/HDL-cholesterol ratio could be employed as a single risk factor instead of using the total cholesterol and HDL-cholesterol as independent measures to estimate CVD risk. Alternatively, LDL-cholesterol and HDL-cholesterol may be used as independent risk factors, but this approach does not

appear to provide any advantage over simply using total cholesterol and HDL-cholesterol in CVD risk estimations (Wilson, 2013).

1.5.2 Atherosclerosis

Arteriosclerosis is a general term used to describe the hardening, narrowing, and loss of elasticity of the arteries resulting in reduced blood flow. Atherosclerosis is a type of arteriosclerosis caused by an atheromatous plaque. The adjective atheromatous describes substances or processes that cause atherosclerosis. The development of atherosclerosis was described in detail by Linton et al. (2015) from which the following summary of information was extracted.

Elevated levels of LDL-cholesterol and apolipoprotein B (apoB) (the main structural protein of LDL-cholesterol) in the blood serum are the initiators of atherosclerosis. The infiltration of apoB into the artery wall causes an inflammatory response, resulting in foam cell formation, known as the "fatty streak" phase of atherosclerosis. The lipoproteins retained within the artery wall activate the endothelial cells. (The endothelium is a thin layer of squamous cells that lines the interior surface of blood vessels). The activated endothelial cells release adhesion molecules and chemo-attractants leading to the attachment of monocytes (white blood cells) in the sub-endothelial space. Activated endothelial cells also promote the recruitment of other immune cells including dendritic cells, mast cells, and regulatory T cells. The monocytes subsequently differentiate into macrophages, which modify LDL-cholesterol particles, by oxidation and glycation, initiating further inflammatory responses. The uptake and accumulation of oxidatively modified LDL-cholesterol by macrophages initiates a wide range of processes resulting in the development of atheromatous plaque, as follows.

Macrophage foam cell formation and endothelial cell inflammation continue to attract more monocytes and immune cells into the sub-endothelial space. The fatty streaks subsequently develop into a fibrous fatty lesion into which smooth muscle cells and extracellular lipids are recruited. As the volume of the lesion enlarges, a stable plaque arises, and the symptoms of atherosclerosis continue to develop. The stable plaque consists of a fibrous cap composed of layers of smooth muscle cells enclosing a matrix network of collagen, proteoglycans, and elastin. The core of a stable plaque also contains macrophage foam cells debris and extracellular lipid resulting from necrosis. Initially the fibrous cap creates a barrier that prevents the rupture of the lesion. Ultimately, as a consequence of necrotic cell death, a decrease in extracellular matrix production, and collagen degradation, a stable plaque develops into a vulnerable plaque, with increased risk of rupture. The rupture of a vulnerable plaque results in the exposure of the core of the plaque to platelets and procoagulant factors in the blood, thereby causing formation of a blood clot (thrombus). Thrombus formation at the site of plaque rupture is responsible for clinical ischemic cardiovascular events, including myocardial infarction, unstable angina, sudden cardiac death, and stroke.

1.5.3 Hypertension

Hypertension (or high blood pressure) is a multifactorial disorder defined as a combination of excessively high levels of systolic and diastolic blood pressure, with mechanistic connections with other disorders including CVD and diabetes (Grundy et al. 1999). Systolic blood pressure is the maximum pressure in the arteries when the heart contracts. Diastolic blood pressure is the minimum pressure in the arteries between the heart's contractions. The symptoms of hypertension are a systolic blood pressure ≥ 140 mm Hg and/or a diastolic blood pressure ≥ 90 mm Hg. The prevalence

of hypertension in people over 16 years old in UK is 31.5% in men and 29.0% in women (British Hypertension Society, 2014).

An important risk factor that results in hypertension is cigarette smoking, which exerts a hypertensive effect, through the stimulation of the sympathetic nervous system (Virdis et al. 2010). Numerous studies have also indicated that dietary intake of salt (sodium chloride) is linked to hypertension, and that that reduction in dietary salt intake may lower blood pressure (Frisoli et al, 2010). Sowers et al. (2001) related the prevalence of hypertension to the high prevalence of CVD. Hypertension causes clogging and weakening of the blood vessels, and can lead to atherosclerosis and narrowing of the arteries, increasing the risk of thrombosis. Arterial damage can also create weak spots that rupture or balloon into the artery wall resulting in an aneurism. Patients with hypertension often present comorbities that are independent risk factors for CVD including obesity, atherosclerosis, endothelial dysfunction, platelet hyper-aggregability, and blood coagulation abnormalities.

The Framingham study included a 30-year follow-up study of normotensive and hypertensive individuals revealing insights into the importance of pulse and blood pressure as markers of hardening of the arteries (Franklin & Wong, 2013). Models of systolic and diastolic blood pressure and pulse pressure significantly predicted CVD events. The Framingham highlighted hypertension as a leading contributor to CVD mortality. Hypertension was also identified globally as a very important risk factor for CVD by the World Heart Foundation (2015). Furthermore, several researchers report a positive association between diabetes, hypertension, and CVD (Grundy et al. 1999). The next section discusses this relationship.

1.5.4 Diabetes

Diabetes is a family of diseases identified by high blood sugar (glucose)

because the body cannot produce sufficient insulin or is unable to use insulin effectively. Prediabetes, also termed impaired glucose tolerance or impaired fasting glucose, is classified by a blood glucose level that is elevated beyond normal levels but not elevated enough to warrant a diagnosis of diabetes. According to Diabetes UK (2016) 4.05 million people are diagnosed with diabetes in the UK, an increase of 119,965 compared to the previous year, and an increase of 65% in the last decade.

A substantial amount of research based epidemiological and pathological evidence suggests that diabetes is an independent risk factor for CVD in both men and women, but all of the major CVD risk factors, including smoking, hypertension, and high LDL-cholesterol also continue to act as independent contributors to the risk of CVD in diabetics. (Grundy et al. 1999). Grundy et al. (p. 1134) argued that "from the point of view of cardiovascular medicine, it may be appropriate to say, diabetes is a cardiovascular disease." The most common forms of diabetes are Type I and Type II. Type I diabetes (also known as insulin-dependent diabetes mellitus, juvenile onset diabetes, or childhood diabetes) is typically diagnosed in children and young adults and accounts for approximately 5% of diagnosed cases. Type II diabetes (also known as noninsulin-dependent diabetes mellitus or adult-onset diabetes) is typically diagnosed in adults over the age of 40, and accounts for the majority (90% to 95%) of the diagnosed cases worldwide. (World Health Organization, 2012b). Type II diabetes, however, is not the cause of death in most diabetics. CVD is listed as the major cause of death in about 65% of persons with Type II diabetes (Wilson, 1998). According to Giugliano et al. (1995) accelerated atherosclerosis is the leading cause of mortality in diabetics.

The Framingham study observed abnormal lipoprotein cholesterol levels in participants with Type II diabetes. Almost all of the diabetic patients had Type II

diabetes, and these patients were much more likely than nondiabetic participants to have low levels of HDL-cholesterol (Winter, 2013). Furthermore, a clustering of metabolic risk factors, called the metabolic syndrome, occurs commonly in patients with Type II diabetes. Metabolic syndrome among patients who went on to develop CVD tended to have low HDL-cholesterol, elevated blood pressure, and impaired fasting glucose. Metabolic syndrome accelerates the formation of glycosylated proteins and advanced glycation products and increases endothelial dysfunction (Hammes et al. 1996). These complications of hyperglycaemia are associated with the causes of CVD in diabetics. According to the National Cholesterol Education Programme's (2001) guidelines, diagnosis of metabolic syndrome is a practical way to identify persons at high risk to develop CVD.

1.5.5 Obesity

Obesity is generally defined as body mass index (BMI) greater than or equal to 30 kg/m^2. The prevalence of obesity has increased globally in all countries since the 1970's (Gortmaker et al. 2011). According to the World Health Organisation (2012a), one in ten adults in the world is obese, and obesity is the fifth leading cause of death in the world, equating to nearly 3 million deaths per year. Kouris-Bazos & Wahlqvist (2007) suggested that if the global obesity crisis is not reversed by 2020, then one third of the adult population worldwide will be obese. Data on increasing obesity rates support the conclusion that obesity has surpassed tobacco use as the leading preventable disease burden in many regions throughout the world (Sassi et al. 2009). The high prevalence of obesity has caused many governments to begin working on measures to halt or reverse the obesity crisis through sustained prevention measures (Swinburn et al. 2011).

Over thirty years ago, the correlation between obesity and cholesterol was

identified as a major risk factor for CVD in the Framingham study in the USA. A longitudinal study was conducted over a period of eight years, with men and women who were 25 to 34 years old at baseline (Wilson et al. 1997). As the body weights of the participants increased, HDL-cholesterol decreased, whilst LDL-cholesterol and very low-density lipoprotein (VLDL) cholesterol increased in both sexes. High BMI was positively correlated with elevated LDL-cholesterol, and negatively correlated with HDL-cholesterol.

A more recent longitudinal study on the correlation between BMI and CVD (specifically coronary heart disease) was conducted by Canoy et al. (2013) as part of The Million Women Study. This cohort study took place between 1996 and 2001 in England and Scotland, with over one million women participants who attended the National Health Service (NHS) breast cancer screening programme. The population-based sample consisted of 1.2 million women (mean age = 56 years) without heart disease or stroke at baseline. The women were followed up prospectively for an average of nine years. The adjusted relative risks increased by 1.23 for every 5 kg/m^2 increase in BMI. Even small increments in BMI among non-obese women were associated with increasing incidence of CVD. The increased risk in proportion to an increase in BMI was consistently identified in various sub-groups of the population, defined by their age, smoking, physical activity, alcohol consumption, and socio-economic class. Even among non-obese women, who were non-smokers, and non-drinkers of alcohol, the risk increased in proportion to a small increase in BMI. Consequently, small shifts in the population distribution of BMI in England and Scotland could potentially have a large effect on reducing the burden of heart disease in the population.

Chen et al. (2013) evaluated the association between BMI and mortality from

CVD among adults in Asia (China, Taiwan, Singapore, Japan, Korea, India and Bangladesh). The relationship between BMI and mortality was U shaped. Increased risk of death from CVD occurred at lower and higher ranges of BMI.

Kromhout et al. (2002) conducted a longitudinal study across male participants in seven countries (called the "seven countries study") which concluded that fluctuating weight in men (the so-called "yo-yo" effect) rather than BMI per se, was associated with greater CVD risk. Men were defined as "fluctuating" when their weight at each examination differed more than 2 kg with their weight at previous or subsequent examinations. This study of body weight from baseline for the following 15 years, concluded that men who gained more than 2 kg, and put on an average weight of 7 kg between examinations, had a 20% greater CVD mortality risk compared to those whose weight remained stable. A similar result, however, was obtained in men who decreased more than 2 kg weight and lost on average 5 kg between examinations.

Obesity in childhood and adolescence is also a serious area of concern for public health. The number of overweight and obese children and adolescents has doubled in the last two decades in the Western world (Ogden et al. 2002; 2010). Several researchers have reported that childhood obesity is also becoming a public health problem in developing countries in Asia and South America (Azfal & Naveed, 2004; Daliri et al. 2013, Perichart-Perera et al. 2007; Raj, 2012). Complications of childhood obesity, such as hypertension continue into adulthood, in the form of CVD risk factors. The significant correlation between childhood obesity and blood pressure level is reported to be a predictor of CVD in adulthood (Lauer et al. 1991; Charakida et al. 2012). In addition to high blood pressure, obese and overweight children may have an abnormal lipoprotein profile including high LDL-cholesterol and total

cholesterol compared to normal weight children. (Daliri et al. 2013; De Onis et al. 2012). Furthermore, Li et al. (2012) found a correlation between the carotid artery thickness and LDL-cholesterol as a CVD risk factor amongst obese children and adolescents, whilst Sinha et al. (2002) reported a high rate of impaired glucose tolerance test and insulin resistance among obese children.

In conclusion, the findings of all of the research outlined above indicate that obese and overweight children and adolescents, as well as adults, are potentially more susceptible to cardiovascular complications and higher risk of CVD morbidity and mortality.

1.6 Lifestyle risk factors for CVD

The four most important risk factors for CVD associated with lifestyle include an unhealthy diet, a low level of physical activity, excessive consumption of alcohol, and smoking tobacco. First, an unhealthy diet, defined as the consumption of modern convenience foods, or junk foods, that are rich in saturated and trans-unsaturated fats, extrinsic sugars, and salt (Crawford, 2013). Second, a low level of physical activity. Moderate physical activity incorporating walking and aerobics may be effective in reducing the risk of CVD (Ross et al. 2009; Teixeira-Lemos, 2012). Third, excessive consumption of alcohol. Several studies have revealed that regular excessive alcohol consumption is significantly correlated with higher levels of LDL-cholesterol, thereby increasing the risk of CVD among heavy drinkers (Mukamal et al. 2001). Fourth, smoking tobacco. The most important smoking-related risk factors for CVD include impairment of endothelial function, arterial stiffness and inflammation, lipid modification, and an alteration of blood factors leading to thrombosis (Virdis, 2010).

1.6.1 Diet

Crawford (2013, p. 67) in a review of the relationship between diet, cancer, and heart disease asserted that:

> "The modern Western diet bears little resemblance to the diet which forged the human genome over many million years. It is operating to distort biology, from even before conception through into late years, with the epidemic of obesity and diabetes likely to lead to stroke and heart disease."

The implications of this assertion are that many people, particularly in the economically advanced regions of Europe and the USA, are currently suffering from the deleterious consequences of an unhealthy diet. An unhealthy diet includes the consumption of modern convenience foods, or junk foods, that are rich in saturated and trans-unsaturated fats, extrinsic sugars, and salt.

Fats consist of long hydrocarbon chains, which can be unsaturated (with double bonds) or saturated, (with no double bonds). Monounsaturated and polyunsaturated fats are an important dietary constituent because they provide essential fatty acids and fat- soluble vitamins. Saturated fats are typically solid at room temperature, and occur naturally in many foods that come from animal sources, including beef, lamb, pork, lard, cream, cheese, and other dairy products. In addition, many baked goods and fried foods contain high levels of saturated fats. Many prepared foods are high in saturated fat content, such as pizza, dairy desserts, and sausages. (American Heart Association, 2014). According to the National Health Service (2012) "Most people in the UK eat too much saturated fat".

Meta-analyses have revealed a significant correlation between dietary saturated fat and serum cholesterol levels (Clarke et al. 1997). The consumption of saturated fat results in an increase in total cholesterol and LDL-cholesterol, compared to the consumption of carbohydrate, polyunsaturated fatty acids, and monounsaturated fatty acids. The effect of dietary saturated fat on the risk of CVD is, however,

controversial. In the UK, the National Health Service (2012) the British Dietetic Association (2012) and the British Heart Foundation (2015) among other health authorities, including the World Health Organization (2015) advise that saturated fat is a risk factor for CVD, and therefore its consumption should be reduced. Some studies contradict this advice. For example, Skeaff & Miller (2009) in a summary of evidence from prospective cohort and randomized controlled trials reported that the intake of saturated fat was not significantly associated with CVD mortality. Siri-Tarino (2010) conducted a meta-analysis of epidemiologic studies and concluded that there was no significant evidence for concluding that dietary saturated fat is associated with an elevated risk of CVD. De Souza et al. (2015) conducted a systematic review of prospective studies related to the intake of saturated fat. The evidence was extremely heterogeneous with many methodological limitations limiting the conclusions. No significant correlations between dietary saturated fat and health outcomes were found in studies where saturated fat replaced refined carbohydrates. Saturated fats were not associated with all causes of mortality or CVD.

In nature, fatty acids with double bonds generally have the cis- as opposed to the trans- isomeric configurations. Trans-fats, or trans-unsaturated fatty acids (tFA) are uncommon in nature, but are produced industrially by adding hydrogen to vegetable oil, which causes the oil to become solid at room temperature. Food is made with hydrogenated oil in order to prolong its shelf life. tFA is found in cakes, biscuits, pies, pizza crusts, crackers, potato, corn, and tortilla crisps, and popcorn. Foods that require deep fat frying contain tFA from the oil used in the cooking process, including potato chips doughnuts and fried chicken Non-dairy coffee creamer and margarines also contain partially hydrogenated vegetable oils. (Martin et al. 2007).

The consumption of tFA been shown to be consistently associated with elevated risk of CVD by raising levels of the LDL-cholesterol, lowering levels of HDL-cholesterol, increasing triglycerides in the bloodstream and promoting systemic inflammation (Brouwer et al. 2010). The major evidence for the effect of trans- fat on CVD comes from the Nurses' Health Study, a longitudinal study that followed a cohort of 120,000 female nurses in the USA for 20 years (Oh et al. 2005). A nurse's risk of coronary heart disease approximately doubled for each 2% increase in trans-fat calories consumed. The systematic review of De Souza (2015) also confirmed that consumption of tFA is positively associated with the risk of CHD mortality.

A diet containing fish is recommended because, unlike meat and poultry products, fish does not contain high levels of saturated fat or trans- fat (American Heart Association, 2015). This recommendation was originally supported by the study of Kromhout et al. (1995) who reported an inverse correlation between fish consumption and 20□year mortality from CVD. Oily fish like salmon, mackerel, herring, trout, sardines and tuna are high in omega-3 fatty acids. Omega-3 fatty acids may prevent CVD by decreasing cardiac arrhythmia, thrombic tendencies, and endothelial dysfunction (Hu & Willett, 2002), although the evidence for this process is not clear.

Sugars are the next component of the diet constituting a potential risk factor for CVD. Dietary sugars have no nutritional value other than to provide calories. Intrinsic sugars are an integral constituent of natural food products include the monosaccharide fructose, found in fruits and vegetables. An unhealthy diet includes extrinsic or added sugars, referring to refined sugars incorporated into many foods such as sweets, biscuits, cakes, pastries, breakfast cereals, and beverages such as soft

drinks. Added sugars include disaccharides, such as sucrose, industrially extracted from sugar cane and sugar beets, (Howard & Wylie-Rosett, 2002).

A literature search revealed that only one long-term clinical trial directly related added sugar consumption to the risk of CVD. The reason for the paucity of research in this area is probably because longitudinal cohort studies relating sugar consumption to CVD are confounded by many other CVD risk factors, that cannot be so easily controlled. Yang et al. (2014) conducted a prospective cohort study of a nationally representative sample of adults in the USA to examine the time trends of added sugar consumption as percentage of daily calories, and to investigate the association between added sugar consumption and CVD mortality. The data were collected in the National Health and Nutrition Examination Survey, with a time trend analysis for 11,722 adults between 1988 and 2010. The mean percentage of daily calories from added sugar increased from 15.7% per individual in 1988-1994 to 16.8% in 1999-2004 but decreased to 14.9% in 2005-2010. Comparing participants who consumed 10.0% to or more calories from added sugar with those who consumed less than 10.0% of calories from added sugar, the hazard risk ratios for CVD were 1.30 and 2.75 respectively, after controlling for age, sex, and ethnicity.

The biochemical mechanisms underlying the association between added sugar intake and CVD risk are not completely understood. Several short-term studies have reported an inverse correlation between dietary sucrose and HDL-cholesterol (Archer et al. 1998; Earnst et al. 1980; Johnson et al. 2009). A diet high in sucrose has been associated with elevated hepatic secretions, which impair the clearance of LDL-cholesterol (Frayn & Kingman, 1995; Parks & Hellerstein, 2000). Because obesity is a definite cause of cardiovascular morbidity and mortality (Eckel et al. 1998) the impact of dietary sugar on weight gain is an important CVD risk factor. Research has

shown that obese people have a significantly higher taste preference for added sugar (Drewnowski et al. 1992); and BMI has been found to be significantly positively correlated with the consumption of added sugar (Klesges et al. 1992). Sugar sweetened beverages are considered to be a major causal factor responsible for the increasing prevalence of childhood, adolescent, and adult obesity (Bachman et al. 2006; Bawa, 2006; Gill et al. 2006; Malik et al. 2006).

Salt (sodium chloride) is another dietary constituent considered in this review as a risk factor for CVD. Many epidemiologic, clinical, and experimental studies, have linked dietary salt intake to hypertension, and a reduction in dietary salt intake has been reported to lower blood pressure (Frisoli et al. 2012). Depending on the baseline blood pressure and degree of salt intake reduction, the systolic blood pressure can be lowered by 4 to 8 mm Hg. When combined with other dietary improvements and lifestyle interventions an even greater decrease in blood pressure can be achieved.

The conclusion that reduction in salt intake reduces hypertension is not, however, definitive. Dumler (2009) conducted a systematic review of long-term controlled randomized trials, which reported that a reduced dietary salt intake was associated with only small decreases (1.1 mm Hg) in systolic but not diastolic blood pressure. Dumler concluded that the long-term impact of reduced salt intake on blood pressure, mortality, and morbidity remains to be defined.

A final dietary factor suggested to be a risk factor for CVD is the hardness or softness of the water that people drink. Hard water contains a high concentration of calcium and magnesium ions, whereas soft water contains a low concentration of these ions. For example, the high incidence of CVD in Scotland may be related to the drinking of soft water, relative to the South of England where the water is harder (Wilson, 2013). Analytical studies, however, have provided little evidence to suggest

that cardiovascular risks are associated with water hardness or calcium levels (Monarca et al. 2006). Although, there is some evidence from epidemiological studies to suggest that there is a significant correlation between the drinking of hard water and lower risk of cardiovascular mortality there is no definitive statistical evidence to prove causality (Lake et al. 2010; Morris et al. 2008; Sengupta et al. 2013).

1.6.2 Regional variations in diet

The consumption of unhealthy foods is not consistent across different geographic regions, and is related to social deprivation and low socio-economic status. Socio-economic differentials may explain disparities between the prevalence of CVD across different regions of the UK. For example, studies in a socially deprived community in the East End of London revealed that about 30% of the calorific value of the food eaten by pregnant mothers came from convenience foods (Doyle et al. 1982). In another study, the diets of school children in the East End of London indicated that a high proportion of their food came from convenience foods (Doyle et al 1994). A recent survey in Scotland (Levin, 2014) revealed that adolescents living in remote rural areas had the highest frequency of consumption of healthy food (fruit and vegetables) and the lowest frequency of consumption of unhealthy food (sweets and crisps). The poorest diet of adolescents was not, however, found in the major cities of Scotland (Glasgow, Edinburgh, and Aberdeen) which are reported to have the highest prevalence of CVD, but in other socially deprived urban areas of Scotland.

A survey conducted among children (age up to 17 years) in Scotland revealed that the mean intake of extrinsic sugars and saturated fat was significantly higher than the UK recommended population average (McNeill et al. 2010). There was, however, no significant correlation between the intake of saturated fat and socio-economic status. Modelling of the data showed that excluding sugar-sweetened soft drinks from

the diet and increasing fruit and vegetable intake by 50% would not restore the intake of extrinsic sugars and saturate fat to recommended levels

Rugg-Gunn et al. (2007) reported that the consumption of extrinsic sugars among English children (age 11-12 years) in the north of England in the year 2000 was substantially higher than recommended, and there was little change over 20 years. Continued and coordinated efforts were recommended at a national, community and individual level to reduce the intake of extrinsic sugars.

Wrieden et al. (2013, p. 1892) highlighted that "the food and nutrient intake of a nation is important for informing the design and evaluation of policy". The researchers conducted a longitudinal survey of dietary change in Scotland between 2001 and 2009, and reported limited improvements, with a small increase in the mean consumption of fruit and vegetables, equating to an increase of < 3 g/person per year. There was also a small decrease in the percentage of food energy from extrinsic sugars and saturated fat concurrent with a reduction in the consumption of whole milk and soft drinks. The authors suggested that greater and more rapid improvements in diet are needed to achieve a significant impact on the health of the Scottish population. The survey conducted by Levin et al. (2014) revealed that the diet of adolescents in Scotland appears to have improved in the last decade. They reported that fruit and vegetable consumption increased between 2002 and 2010, while consumption of sweets, chips and crisps also fell. The overall healthy eating score increased significantly during this period. Fruit and vegetable consumption was, however, most frequent among children with higher levels of socioeconomic status.

Fleming et al. (2013) reviewed the available evidence to show that the dietary patterns of populations with low levels of CVD and high longevity have similar characteristics, specifically that diets are higher in plant foods and lower in animal

products. The benefits of this dietary pattern are recognized in the Mediterranean Island of Sardinia, which is characterized by exceptional male longevity. The Mediterranean diet includes a high intake of monounsaturated fat, plant proteins, whole grain breads, cereals, pasta, moderate alcohol and low intake of red meat, refined grains and sweets. The benefits of a Mediterranean diet among persons with high cardiovascular risk was confirmed by Estruch et al. (2013) in a randomized controlled trial involving a total of 7447 persons for five years. The incidence of cardiovascular events among the two groups assigned to the Mediterranean-diets were significantly lower than among the control group.

1.6.3 Consumption of alcohol

The Framingham study revealed that regular excessive alcohol consumption was significantly correlated with lower levels of HDL-cholesterol, and higher levels of LDL-cholesterol, thereby increasing the risk of CVD among heavy drinkers (Mukamal et al. 2001). Obesity has a moderating effect on the relationship between CVD and consumption of alcohol. Canoy et al. (2013) found that the risk of CVD was higher among female alcohol drinkers in England and Scotland with a BMI $\geq$ 35 kg/m^2 than that for female drinkers with a BMI < 25 kg/m^2.

The effects of the consumption of alcohol on CVD risk are regional. Disproportionately high levels of binge drinking (i.e., the consumption of an excessive amount of alcohol in a short time period) in Scotland may be one of the potential causes of the inequalities reported in CVD prevalence across the UK (Bhatangar et al. 2014).

1.6.4 Smoking

For over 25 years, governments have implemented policies to warn citizens about the damaging effects of smoking tobacco, particularly cigarettes, because

smoking is a well-established risk factor for hypertension, atherosclerosis, coronary heart disease, and peripheral artery disease (Wilhemson, 1988). Nicotine increases the oxygen requirement of tissues and organs, including the heart, by increasing the level of fatty acids in the blood, thereby increasing the risk of CVD. Cell repair processes in the body are impaired because smoking depresses antioxidant levels, causing oxidative stress, so that smokers have an elevated risk of early development of CVD (Bhatt, 2003). Furthermore, smoking promotes damage to blood vessels by altering the lipid content of the blood resulting in impaired circulation and thrombosis (Mojos, 1998). HLD-cholesterol levels are reduced in smokers, whilst total cholesterol and LDL-levels are increased, elevating the risk of CVD. The Framingham study revealed that on average, compared with non-smokers, cigarette smoking was associated with HDL-cholesterol levels that were approximately 4 mg/dl lower in men and 6 mg/dl lower in women (Garrison et al. 1978; Wilson, 2013). Virdis et al. (2010) found that the most important smoking-related risk factors for CVD are impairment of endothelial function, arterial stiffness and inflammation, lipid modification, and an alteration of blood factors leading to thrombosis. The levels of plasma fibrinogen and platelet counts, which are early stage markers of CVD, have also found to be higher among smokers than among non-smokers (Kannan et al. 2013).

Primatesta et al. (2001) used data from the annual Health Survey for England (1994 to 1996) to investigate the difference in blood pressure between smokers and non-smokers in a nationally representative sample. The results indicate a difference of 2 mm Hg systolic blood pressure between smokers and non-smokers. The findings indicated complex interrelations between gender, smoking, alcohol intake, and BMI. Among men, a significant interaction was found between obesity and the association between smoking and blood pressure. In women, blood pressure differences between

non-smokers and smokers were significant between those who did not and those who did drink alcohol. The effects of smoking on CVD risk appear to be regional in the UK. Disproportionately high levels of cigarette smoking in Scotland may be one of the potential causes of the inequalities reported in CVD prevalence across the UK (Bhatangar et al. 2014; Popham, 2011).

1.6.5 Physical activity

The Framingham study showed that physical activity, especially activities associated with aerobic conditioning, were associated with increased HDL-cholesterol levels in both men and women, thereby reducing the risk of CVD (Wilson, 2013). Increased understanding of the importance of physical activity as a risk factor for CVD comes mainly from intervention studies. Physical exercise is recommended by clinicians as beneficial to reduce the onset of CVD and comorbidities. Physical exercise incorporating walking and aerobics can be effective in reducing the risk of both Type II diabetes and CVD (Ross et al. 2009; Teixeira-Lemos, 2012). Gill and Cooper (2008) reviewed 20 studies concluding that moderate physical activity for approximately 150 minutes per week can have beneficial effects. Several other studies have demonstrated that interventions incorporating both physical activity and improved dietary practices help to lower blood glucose levels, thereby helping reduce the likelihood of developing Type II diabetes as well as CVD (Auchincloss et al. 2009). Freak-Poli et al. (2010) assessed the prevalence of risk factors for CVD and diabetes Type II diabetes among 762 employees with sedentary occupations. The majority of employees were not meeting recommended guidelines for physical activity and a substantial proportion were unaware of their increased risk. The effects of physical activity on CVD risk appear to be regional. Disproportionately low levels

of physical activity in Scotland may be one of the potential causes of the inequalities reported in CVD prevalence across the UK (Bhatangar et al. 2014).

1.7 Tracking CVD risk factors from childhood into adulthood

Several cohort studies have tracked CVD risk factors from childhood to adulthood. All these studies have concluded that exposure to risk factors early in life influence the development of CVD in adults (Arts et al. 2014). Early longitudinal studies indicated that the severity of the atherosclerotic lesions among young adults were associated with high levels of total cholesterol, blood pressure, BMI, and cigarette smoking (Lauer et al. 1975). Subsequently, Stary (1989) observed the onset and progression of atherosclerosis in over 3000 individuals aged 15 to 34 years. Atherosclerotic lesions were found in more than half of the teenagers, and these lesions progressed into clinically significant lesions by young adulthood.

More recently, Juonala et al. (2005) found that as many as 10–20% of young adults may have atherosclerotic lesions. Mikkila et al. (2005) analysed longitudinal data collected from a cohort to demonstrate that the association between risk factors in childhood and risk factors measured 27 years later were strongest for total cholesterol and LDL-cholesterol. Dwyer et al. (2013) measured high levels of LDL-cholesterol, blood pressure, and obesity among a cohort of school children that were linked to the development of atherosclerosis in young adulthood. The findings of cohort studies are critical with respect to prevention of CVD because young people at risk of developing atherosclerosis could potentially be identified and treated decades before clinical manifestation of the disease.

Zieske et al. (2002) concluded that the risk profile for CVD was reduced among young people who had a healthy lifestyle, including being physically active, and consuming a diet low in saturated fatty acids and sodium. All the evidence

indicates that unhealthy dietary choices among young people are a major determinant of the risk of CVD. Because dietary patterns established early in life carry into adulthood and are strongly associated with risk of CVD, interventions to promote positive dietary changes are essential for adolescents with poor dietary choices entering adulthood (Arts et al. 2014; Biswasit et al. 2015; Gordon-Larsen et al. 2004; McDade et al. 2011; Slining et al. 2013). The following section presents more detailed information on the prevention of CVD.

1.8 Prevention of cardiovascular disease

Maier (2014) suggested that about half of all adults in the UK have at least one CVD risk factor. Unal et al. (2010) estimated that between the years 1981 and 2000, 58% of the decline in the death rate from CHD in the UK was due to the control of risk factors. Hotchkiss et al. (2014) suggested that substantial contributions from population reductions in high blood pressure, obesity, and other CVD risk factors have already diminished the adverse trends for the higher prevalence of CVD in Scotland. Further population-wide interventions were recommended to reduce CVD mortality and inequalities in Scotland.

A longitudinal survey (Hotchkiss et al. 2014) revealed that improved treatments accounted for less than half (43%) of the fall in mortality due to CVD in Scotland between 2000 and 2010. The most important treatments were for hyperlipidemia (13%), other prevention drugs (11%), and chronic angina (7%). Risk factor improvements accounted for approximately 39% of the decline in mortality from CVD. Control of hypertension contributed to over one third (37%) of the decline in mortality. Smaller contributions came from reducing levels of total cholesterol (9%), smoking (4%), and inactivity (2%). However, increases in obesity and diabetes

offset some of the benefits of other preventative measures, increasing mortality by 4% and 8% respectively.

Socio-economic differences do not directly explain why the mortality due to CVD varies across different regions of the UK. The effects of social-economic differences between Scotland and England are confounded by regional patterns in unhealthy behaviours, such as smoking, unhealthy diet, alcohol consumption, and physical inactivity (Whitley, et al. 2014).

Because obesity is a risk factor for CVD, dietary changes are recommended to maintain weight reduction in obese persons in the USA (Eckel & Krauss, 1998) and Europe (Ruxton et al. 1999; Gortmaker et al. 2011) including Scotland (Castle, 2015). There is evidence to support these recommendations, because diets low in extrinsic sugar have been associated with weight loss (Colditz et al. 1990) and therefore an important strategy to reduce CVD risk limit consumption of foods that are high in added sugar. Improving the diet of the Scottish population has been a government focus in recent years (Levin, 2014: Levin et al. 2014). The Schools (Health Promotion and Nutrition, Scotland) Act (2007) imposes duties on Scottish Ministers, education authorities, and managers to endeavour to ensure that public and grant-aided schools are health promoting. The act includes regulations to ensure that all food and drink provided in schools complies with specified nutritional requirements. To date, however, the NHS in Scotland has not published official guidelines aiming to reduce the risks of CVD among university students through improvements in diet, even though there is a considerable body of information in the literature to demonstrate that a healthy diet can reduce the risks of CVD.

Nutritional authorities including the UK Scientific Advisory Commission on Nutrition (2007) advise that consumption of saturated fat and trans-fat should be

reduced. There is evidence to indicate that reduction in the intake of saturated fat may reduce the risk of CVD. Micha & Mozaffarian (2010) and Schwab (2014) reviewed the available evidence to conclude that the risk of CVD was reduced if consumption of saturated fat was replaced with polyunsaturated fat. Hooper (2011) also found that reducing saturated fat in the diet reduced the risk of cardiovascular events. De Souza (2015) confirmed that reduced consumption of trans- fats lowers the risk of CHD mortality.

Frivoli et al. (2012) recommended that a reduction in dietary salt intake could delay or prevent hypertension and is one of the simplest cost-saving methods to reduce CVD morbidity and mortality. The Heart Foundation (2007) recommended that for the general population, sodium intake should be reduced to < 2300 mg/day, and for patients at risk of CVD, sodium intake should be < 1550 mg/day. An increase in the intake of dietary fibre decreases LDL-cholesterol. Dietary fibre also has a role in glycaemic control and in the prevention of weight gain. Although these effects are modest, they may play a role in the apparent protective effect of dietary fibre on the risk of CVD (Bazzano et al. 2003). There is ample evidence in the literature to show that CVD is preventable by dietary patterns that include the consumption of non-hydrogenated unsaturated fats as the predominant form of dietary fat, whole grains as the main form of carbohydrate, together with an abundance of fruit and vegetables, and adequate omega-3 fatty acids (Hu & Willett, 2002).

Healthy dietary patterns, must however, be combined with regular physical activity, avoidance of tobacco smoke, and maintaining a healthy body weight. Based on the clinical evidence, various organizations have produced guidelines for the prevention of CVD. For example, the World Health Organization (2015b) guidelines recommend that eating healthy foods, including at least five servings of fruit and

vegetables a day, taking regular exercise, and limiting salt intake to less than one teaspoon a day will help to prevent CVD. In the USA, the American Heart Association (AHA) has provided formal evidence based clinical guidelines for CVD prevention for individuals of 20 years old and over (Matzo, 2008; Mosco et al. 2007). The evidence, based on 246 randomized clinical trials and prospective cohort studies, indicated that CVD prevention should include the numerous lifestyle interventions. These include: (a) a weight management programme to ensure a BMI of 18.5 to 24.9 kg/m^2; (b) the cessation of smoking and the avoidance of environmental tobacco smoke; (c) moderate daily physical activity, such as brisk walking to sustain a loss in weight; (d) a diet rich in fruits and vegetables, and high in dietary fibre, and fish at least twice weekly; (e) the use of dietary supplements, including 850 to 1000 mg of omega-3 fatty acid; and (f) treatment for depression, if required. The AHA guidelines appreciated that lifestyle habits are difficult to implement in practice, and therefore recommend that nurses and clinicians should take every opportunity to discuss the implementation of preventive lifestyle interventions with their patients into order to maximize the reduction in the risks due to CVD.

1.9 Purpose of this research

The transition period from adolescence to young adulthood is a high-risk period for CVD associated with changes in body weight, entering college, living away from home for the first time, and experiencing increased independence and responsibility for dietary choices. There has, however, been a limited amount of previous research to explore the incidence of multiple CVD risk factors among college and university students (Arts et al. 2014; Biswasit et al. 2015; Cheng et al. 2002). No previous studies have been conducted specifically to examine multiple CVD risk factors among university students in Scotland. In the light of this paucity of

information, the purpose of the current research was to identify the risk factors for CVD among Heriot-Watt university students in Edinburgh, Scotland, and to explore the possible influence of diet and life style on these factors. The overarching research questions that guided the current research were as follows:

Research Question 1: What are the dietary and lifestyle risk factors for cardiovascular disease among university students in Edinburgh, Scotland?

Research Question 2: To what extent can the students be classified into groups according to their risk factors for cardiovascular disease?

Research Question 3: To what extent are the risk factors for cardiovascular disease statistically related to each other?

Answering these questions has a practical significance because a better understanding of CVD risk factors will support collaborative efforts by health care professionals, policy makers, public health administrators, and communities to develop and engage in preventative lifestyle modification programmes that may in the future help to reduce the high prevalence of CVD in the population of Scotland and elsewhere.

1.10 Research design

The design of the current research is described as exploratory, descriptive, and correlational. Exploratory research designs were originally promoted by Tukey (1977) to encourage researchers to search for new relationships and patterns among data, by use of inductive reasoning to generate new hypotheses, rather than by use of deductive reasoning to test predefined hypotheses. Subsequently, Tukey (1980); Holland et al. (1989); and Kline (2004) argued that researchers place too much emphasis on confirmatory data analysis, and more emphasis needs to be placed on

exploratory data analysis. Consequently, this study did not include the testing of hypotheses.

Descriptive research is defined as an attempt to provide additional information about a target population with respect a defined problem. Descriptive research aims to reveal what is happening in more detail, to fill gaps in the literature, and to expand knowledge and understanding (Babbie, 2010). The fundamental characteristics of a descriptive research design are that one or more surveys are conducted to collect information from a target population, without changing their environment, thus participants are not assigned into groups, nor is any part of their environment manipulated. In this study, the target population was undergraduate Heriot-Watt university students in Edinburgh, Scotland. The problem was defined as the risk factors for cardiovascular disease, in particular, diet and aspects of lifestyle.

The research design involved the use of two surveys. The first survey involved the administration of the "Diet and Health Screening Questionnaire (see Appendix A) to 156 students in 2013. The aims of the first survey were to (a) describe the general characteristics of the students in terms of their demographics, health, family history of disease, lifestyle, diet, and consumption of beverages; (b) identify the occurrence of CVD risk factors; and (c) conduct a statistical analysis of the empirical data. The second survey, conducted in 2014, involved recruiting 40 students who had already completed the "Diet and Health Screening Questionnaire". The aims of the second survey were to (a) describe the demographic and lifestyle characteristics of the students; (a) estimate their daily energy and nutrient intake; (b) take blood measurements (plasma concentrations of lipoproteins, insulin, and blood glucose); (c) collect anthropometric measures (BMI, body fat, waist and hip circumference, blood pressure); and (d) conduct a statistical analysis of the empirical data.

Correlational research is defined as exploring the statistical relationships between two or more variables (Creswell, 2014). Accordingly, in the current research, statistical analysis was conducted to determine the extent to which the identified risk factors for CVD were related to each other. The limitation of correlational research is that the statistical analysis of variables collected in a survey do not prove the existence of causal relationships (Pearl, 2009). It was not possible to determine if cause and effect relationships existed between the identified CVD diet and lifestyle risk factors. Nor was it possible to predict the ultimate development of CVD among the students, because the outcomes of the CVD risk factors would probably not develop for many years.

1.11 Theoretical framework

Up to this point, the introduction to this dissertation has considered only the empirical framework that underpins this study, based on a summary of factual information extracted from the literature regarding previous studies. A theoretical framework is also imperative to guide effective research, because every researcher must be able to support, justify, and defend a theoretical approach to the methods that s/he uses to collect, analyse, and interpret empirical data (Creswell, 2014; Gay & Weaver, 2011; Greenwald, 2012). Although the current study was largely empirical, and not exclusively underpinned by any specific theories, its perspective was broadly associated with the philosophy of positivism. Positivism posits that human knowledge is acquired, verified, and developed, by interpreting empirical evidence through reason and logic (Aliyu et al. 2014; Giddens, 1997; Wayhuni, 2012).

For nearly a century, positivism was supported by the confirmatory approach to the collection, analysis, and interpretation of quantitative data. The confirmatory approach requires the testing of predefined null and alternative hypotheses, or

statements proposing that systematic relationships between the variables of interest do not exist, or alternatively, that they do exist (Huberty, 1999). Hypothesis testing is associated with the computation of inferential test statistics and probability (p-values) to indicate so called statistical "significance". Conventionally $p < .05$ indicates that the null hypothesis should be rejected, and that the results should be declared as "significant" implying that they were exceptional, because they were not caused by random chance (Hurlbert & Lombardi, 2009).

The traditional approach to positivism, based on hypothesis testing, which many researchers consider to represent a gold standard in biological and medical research, has been questioned. The importance of this controversial issue, which is currently changing the approaches used by many biological and medical researchers to collect, analyse, and interpret data, was brought to the researcher's attention by an article in *Nature* (Nuzzo, 2014). This article, which highlights the many errors associated with the misuse and misinterpretation of inferential statistic and p-values, was the most highly recommended publication in biology and medical research in 2014 (F1000Prime, 2014). Another recent article in *Nature* (Halsey et al. 2015) further attempted to discourage researchers from following the flawed practice of using p-values, and emphasized the need for effect sizes. All researchers in biology and medical research need to be aware of the issues discussed in these articles, because they have serious implications for future research.

There are many other articles published in the last 20 years criticizing the conventional use of inferential test statistics and p-values. These articles argue that p-values do not: (a) provide any information about the strength of the relationships between variables; (b) prove the existence of causal relationships between variables, or (c) determine whether a hypothesis is true or false (Falk & Greenbaum, 1995;

Gliner et al. 2002; Hubbard & Lindsay, 2008; Huck, 2009; Krueger, 2001; LeCoutre et al. 2001; Nickerson, 2000; Rosnow & Rosenthal, 1989; Schervish,1996; Sotos et al. 2007; Suter,1996; Wagenmakers, 2007). Iaonnidis (2005) went so far as to assert that most published findings in medical research are false, mainly due to the misinterpretation of statistical significance. Several other authors have highlighted the many statistical errors in the medical research literature (e.g., Chia, 1997; Ghazemi, & Zahediasl, 2012; Orlitzky, 2012; Strasak et al. 2007; Taylor et al. 2011, Young, 2007). Fernandes- Zilak & McCloskey (2008, p.1) called the reliance on inferential test statistics and p-values as the "cult of statistical significance" and claimed that this unsound approach has cost justice, jobs, and lives. The researcher agrees with Sedlmeir (2009) that scientists need to be liberated from the mindless testing of hypotheses, and also with Hurlbert and Lombardi (2009) that the classical decision-making framework for testing hypotheses using p-values, despite its prevalence for nearly a century, has collapsed, and should no longer be taught to students, or applied by researchers. Therefore, no hypotheses were formulated or tested in the current study. Following the many arguments in the literature, and the modern trend of informed researchers, the theoretical stance of the researcher was not to compute p-values in order to test hypotheses concerning the relationships among the risk factors for CVD.

1.12 Ethical considerations

The researcher complied with the Heriot-Watt ethical review process. The research was approved by the University and School ethics committees. All of the students gave their informed consent to participate in the surveys after the researcher had provided them with details of the nature and purpose of the research. The rights of the participants to confidentiality and anonymity were protected. Only code numbers

and not names were used to identify the respondents in the data files. No data that could be used to identify any of the participants was shared. The researcher protected the confidential information provided by the respondents throughout this study. It was possible that some participants could feel disturbed about providing personal data in the public domain. For this reason, the survey did not include any sensitive questions that might make the participants feel uncomfortable or embarrassed.

1.13 Structure of dissertation

Chapter Two provides: (a) a more detailed review of the existing literature on the risk factors for CVD associated with the diet and lifestyle of college/university students; (b) a description of the administration of the "Diet and Health Screening Questionnaire"; (c) the methods used to analyse the students' responses; (d) a statistical analysis of the results, and (e) a discussion of the results of Chapter Two in the context of the literature.

Chapter Three provides: (a) a description of the methods used to collect and analyse the daily energy/nutrient intake, blood constituents and anthropometric measures of the students; (b) a statistical analysis of the results; and (c) a discussion of the results in the context of the literature. Chapter Four presents the general conclusions, including recommendations for practice and future research.

Chapter Two

Diet and Health Screening Survey

2.1 Introduction

Chapter Two extends the information provided in Chapter One by discussing existing evidence concerning the risk factors for CVD among college/university students. This literature review is followed by a description of the administration of the "Diet and Health Screening Questionnaire" and the methods used to analyse the students' responses to the questionnaire. The results of a statistical analysis of the response data are presented followed by a discussion of the findings in the context of the literature and subsequent research.

2.2 Risk factors for cardiovascular disease among college/university students

A search of the recent literature revealed that few previous studies have been conducted to explore the incidence of CVD risk factors specifically among college and university students. Arts et al. (2014) published a review of the literature on coronary heart disease (CHD) risk factors among college students, citing several sources of empirical data. This review revealed that more than one-half of young adults aged 18–24 years old may have at least one CHD risk factor and nearly one-quarter may have atherosclerotic lesions. The extent of atherosclerosis was correlated with a number of risk factors, including unhealthy diet, which contributed to obesity and hyperlipidemia. The authors argued that the identification of risk factors among young adults is critical in order to promote lifestyle changes that will prevent the progression of CHD. This review established the urgent need for increased screening, risk assessment, education, and management of risk factors for CHD among young adults. The authors concluded that screening efforts should focus on the assessment of all CHD risk factors and lifestyle habits (diet, exercise, and smoking), blood pressure, glucose, and BMI in addition to the traditional cholesterol measures in order to achieve long-term risk reduction.

Subsequently, Biswasit et al. (2015) conducted a survey of 458 medical students with a mean age of 19.4 years by administering a modified version of WHO STEPS (Stepwise approach to chronic disease risk factor surveillance) questionnaire, as well as evaluating the daily intake of fruits and vegetables by each student. The physical activity of the students was "adequate" if they practiced exercise or outdoor sports for at least 30 min/day for more than five days in the past week, but "inadequate" if their physical activity was less than five days per week. Smoking was categorized as "current users'" (i.e., the student used tobacco at least once in the last 30 days preceding the survey) or "never users" (i.e., the student had not used tobacco; even once in the lifetime); however, the survey did not include the students who only had one or two cigarettes and then stopped. Similar definitions were used for alcohol consumption. Anthropometric factors (height and weight) and blood pressure were measured for each student.

The results of the survey indicated that most of the students had very poor compliance with a healthy diet and they exhibited relatively low levels of physical activity. The vast majority of the students (96.7%) consumed less than the recommended daily intake of fruits and vegetables, and most of the students (91.3%) ate convenience foods. Snacking on junk food between meals was reported by 40% of the students. The lifestyle environment of the students, which required them to achieve high levels of academic performance, demanded long hours of sedentary activity. About one-third of the students had no physical exercise at all, and most were not involved in any kind of sports activity (Biswasit et al. 2015).

Biswasit et al. (2015) came to the same conclusion as Arts et al. (2014) recommending an urgent need to intervene to bring about a change in college students' unhealthy behaviour. The authors suggested that a health education

programme with structured dietary practices and exercise regimes should be incorporated into the students' curriculum. This suggestion is feasible in practice, because randomized control trials have confirmed that structured educational programmes have beneficial effects to change the lifestyles of students, resulting in the prevention of the early development of CVDs (Cheng et al. 2003).

The above literature review revealed that the current research on students at Heriot-Watt University, Edinburgh, should focus on screening efforts to measure multiple CVD risk factors, including daily energy/nutrient intake and physical activity, as well as blood and anthropometric measures, providing a rationale and direction for the current study. The first stage of this process was to administer the "Diet and Health Screening Questionnaire" as follows.

2.3 Methods

The "Diet and Health Screening Questionnaire" (see Appendix A) was administered in 2013 to collect self-reported responses from a convenience sample of undergraduate students at Heriot-Watt University. The aim of the survey was to describe the general characteristics of the students in terms of their demographics, health, family history of disease, lifestyle, diet, and consumption of beverages. The responses to the questionnaire provided preliminary information to address Research Question 1: What are the dietary and lifestyle risk factors for cardiovascular disease among university students in Edinburgh, Scotland? and Research Question 3: To what extent are the risk factors statistically related to each other? Of particular interest to this study was the relationship between obesity of young people and the consumption of soft drinks containing extrinsic sugars, which has been established in previous surveys (Bachman et al. 2006; Bawa, 2006; Gill et al. 2006; Malik et al. 2006). The response data collected using the "Diet and Health Screening Questionnaire" were not

applicable to address Research Question 2: To what extent can the students be classified into groups according to cardiovascular risk factors?

2.4. Administration of Diet and Health Screening questionnaire

The target population for this survey consisted of undergraduate students enrolled at Heriot-Watt University, living in Edinburgh, Scotland in 2013-2015. The convenience sample consisted of 156 students who volunteered to provide their responses to the "Diet and Health Screening Questionnaire". The current size of the undergraduate population at Heriot-Watt University is 5796 (https://www.hw.ac.uk) therefore the volunteers represented 156/5796 = 2.7% of the target population.

Students who volunteer to participate in research may provide different data to those who do not agree to participate, resulting in sampling bias (Fraenkel & Wallen, 2010) Therefore, because the sample consisted entirely of volunteers, and was not drawn randomly from the target population, the findings of this study may have limited external validity (i.e., the conclusions may not necessarily be generalizable to all undergraduate students at Heriot-Watt or any other University, but may only be pertinent to the sample of students who volunteered to participate). Furthermore, limited generalizability to the target population is indicated by the sample size calculation illustrated in Figure 2.1.

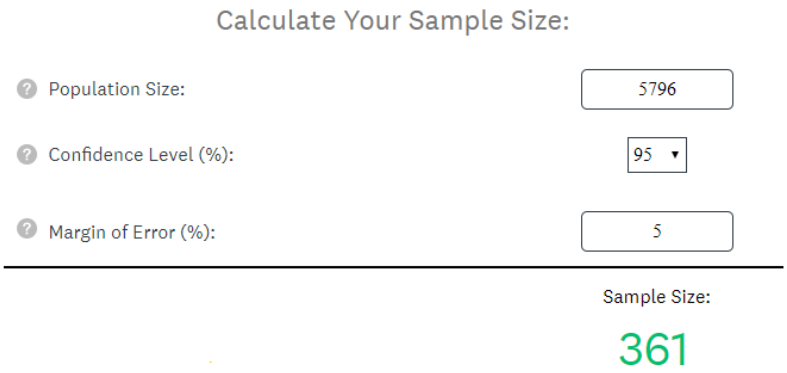

Figure 2.1 Sample size calculation

Source: https://www.surveymonkey.com/mp/sample-size-calculator/

The online calculator predicted that the minimum sample size required for the researcher to be 95% confident that the sample was representative of the population, with a 5% margin of error, should be at least 361 students (i.e., over twice the actual sample size).

2.5 Analysis of responses to questionnaire

The responses of 156 students to the "Diet and Health Screening Questionnaire" were analysed using IBM SPSS vs. 21.0 using the protocols described by Field (2013). The level of measurement of all of the response data was categorical, meaning that the frequencies of the responses to all of the questions were classified into predetermined categories. Therefore, all of the results are presented in the form of the frequency distributions (counts and percentages) of the number of students who responded to each category. This survey did not generate quantitative measures (e.g., responses to questions based on Likert or similar rating scales that were aggregated to operationalize constructs). Consequently, it was not possible to compute descriptive statistics (e.g., mean and standard deviation) or conduct inferential statistics (e.g., t-tests, analysis of variance, and correlation analysis) because these methods are not applicable for categorical data (Agresti, 2013). Furthermore, it was it not possible using categorical data to provide statistical evidence of the validity or reliability of the responses (e.g., using factor analysis and Cronbach's alpha). The frequency distributions of the respondents classified by sex, age, BMI, emotional health, physical health, family history of disease, dietary characteristics, consumption of beverages, and lifestyle characteristics were computed and tabulated.

The associations between the frequencies in each category were measured using Cramer's V coefficients, which is a measure of the strength of the association between two categorical variables in a cross-tabulation,

generating a value between 0 and +1. The stronger the association, then the more Cramer's V tends towards +1. Cramer's V was computed using the following formula:

$$\sqrt{\frac{\chi^2/n}{\min(k-1, r-1)}}$$

Where: Cramer's V is the square root of the Chi-square (χ^2) statistic, divided by the sample size (*n*), multiplied by the smallest dimension in the contingency table (i.e., the number of columns minus one (*k*-1), or the number of rows minus one (r = 1) depending on which was the smallest (Agresti, 2013). Cramer's V is not an inferential statistic, associated with a p-value, but is an effect size, providing an index of the strength of the association between two categorical variables. A p-value (which is a function of the sample size) does not provide any information about the strength of an association. The advantage of Cramer's V is that it factors out the sample size, so that, unlike χ^2, it does not automatically increase in magnitude when the sample size is large; however, because χ^2 values tend to increase with respect to an increase in the number of cells in a cross-tabulation, the greater the difference between the number of rows and columns, then the more likely that Cramer's V tends towards 1.0 without strong evidence of a meaningful association. Another limitation of Cramer's V is that it does not have a sign, so it does not determine the direction of the association (i.e., whether the association is positive or negative). The magnitudes of Cramer's V coefficients indicated the clinical significance of the associations (i.e., their meaningfulness in the context of medical research). Cramer's V < 0.25 represented little or no clinically significant association. Cramer's V between 0.25 and 0.64 represented an association with weak to moderate clinical significance. Cramer's V ≥

0.65 represented a strong association with substantial clinical significance (Ferguson, 2009).

Cross-tabulations were also used to examine the differences between the expected and observed frequencies. The frequencies expected by random chance in the rows and columns of a cross-tabulation were computed using the formula:

$$e_{ij} = \frac{\text{row } i \text{ total} * \text{col } j \text{ total}}{\text{grand total}}$$

Where: the expected frequency (e_{ij}) is the cell count in the i^{th} row and the j^{th} column of the cross-tabulation. The direction and strength of the associations between the categories in the rows and columns of a cross-tabulation were indicated by how far the observed frequencies were away from the expected frequencies (Agresti, 2013).

2.6 Results

The responses to the "Diet and Health Screening Questionnaire" are presented to describe (a) the characteristics of the respondents, with respect to their demographics, health, family history of disease, diet, lifestyle, and consumption of beverages; (b) the associations between the frequencies of consumption of different types of beverage by the respondents; (c) the associations between the frequencies of consumption of different types of beverage by the respondents and their demographic, health, family history of disease, diet, and lifestyle characteristics; and (d) the associations between the diet and lifestyle characteristics of the respondents and their demographic characteristics.

2.6.1 Characteristics of participants

Table 2.1 summarizes the sex and age distributions of the respondents. Females represented over half (n = 88, 56.4%) of the sample. Most of the participants (n = 139, 89.1%) were 17-24 years old, and relatively few (n = 3, 1.9%) were over 34

years old. No data were collected to determine the frequency distributions of racial/ethnic categories among the students.

Table 2.1 Frequency distributions of sex and age

Demographic characteristic	Category	Frequency	Percent
Sex	Female	88	56.4%
	Male	68	43.6%
Age (Years)	17-24	139	89.1%
	25-34	14	9.0%
	35-44	2	1.3%
	45-54	1	0.6%

Table 2.2 presents the health characteristics of the sample Over half of the participants (n = 93, 59.6%) were normal weight, but over a third were classified as overweight (n = 39, 25.0%) or obese (n = 14, 9.0%). The majority described their emotional health as moderately happy (n = 93, 59.6%) or very happy (n = 43, 27.6%). Most of the respondents reported that physically they were moderately healthy (101, 64.7%) or very healthy (n = 29, 18.6%).

Table 2.2 Frequency distributions of health characteristics

Health characteristic	Category	Frequency	Percent
BMI (kg/m^2)	< 18.5 (underweight)	10	6.4%
	18.5-24.9 (normal)	93	59.6%
	25.0-29.9 (overweight)	39	25.0%
	≥ 30 (obese)	14	9.0%
Emotional Health	Very unhappy	2	1.3%
	Moderately unhappy	16	10.3%
	Don't know	2	1.3%
	Moderately happy	93	59.6%
	Very happy	43	27.6%
Physical Health	Very unhealthy	3	1.9%
	Moderately unhealthy	19	12.2%
	Don't know	4	2.6%
	Moderately healthy	101	64.7%
	Very healthy	29	18.6%

Table 2.3 presents the frequency distributions of family history of disease. The family histories of about one half (n = 76, 48.7%) included diabetes, about one quarter (n = 43, 27.6%) included high cholesterol and heart disease/stroke (n = 40, 25.6%)

Table 2.3 Frequency distributions of the family history of disease

Family history of disease	Category	Frequency	Percent
Diabetes	No	76	48.7%
	Yes	72	46.2%
	Don't know	8	5.1%
High cholesterol	No	89	57.1%
	Yes	43	27.6%
	Don't know	24	15.4%
Heart disease/stroke	No	106	67.9%
	Yes	40	25.6%
	Don't know	10	6.4%

Table 2.4 presents the frequency distributions of the self-reported dietary characteristics. A few (n = 9, 5.8%) reported that they were vegetarians, and even less (n = 2, 1.3%) were vegans. Lactose intolerance was also infrequent (n = 10, 6.4%). The majority (n = 94, 60.3%) ate breakfast every day, but only about one third (n = 55, 35.3%) had five servings of fruits/vegetables every day, or took dietary supplements, such as vitamins (n = 53, 34.0%).

Table 2.4 Frequency distributions of dietary characteristics

Dietary characteristic	Category	Frequency	Percent
Vegetarian	No	147	94.2%
	Yes	9	5.8%
Vegan	No	154	98.7%
	Yes	2	1.3%
Lactose intolerant	No	146	93.6%
	Yes	10	6.4%
Breakfast every day	No	62	39.7%
	Yes	94	60.3%
Five servings of fruits/vegetables every day	No	101	64.7%
	Yes	55	35.3%
Dietary supplements	No	103	66.0%
	Yes	53	34.0%

Table 2.5 presents the frequency distributions of lifestyle characteristics.

Table 2.5 Frequency distributions of lifestyle characteristics

Lifestyle characteristic	Category	Frequency	Percent
Smoking	No	139	83.3%
	Yes	26	16.7%
Walk for ten minutes every day	No	7	4.5%
	Yes	149	95.5%
Three strong physical activities/week	No	103	66.0%
	Yes	53	34.0%
Three moderate physical activities/week	No	79	50.6%
	Yes	77	49.4%
Physical activity level	Inactive	2	1.3%
	Less active	31	19.9%
	Moderately active	91	58.3%
	Very active	32	20.5%

Most of the participants did not smoke (n = 139, 83.3%). The majority reported that they walked for ten minutes every day (n = 149, 95.5%); however, only about one third reported that they had three strong physical activities/week (n = 53, 34.0%) and about one half had three moderate physical activities per week (n = 77, 49.4%). Over a half (n = 91, 58.3%) described their lifestyle as moderately active, but about one fifth (n = 33, 21.2%) reported they has an inactive or less active lifestyle. Table 2.6 presents the frequencies of consumption of different beverages. The most frequently consumed drinks (≥ 1 glass/day) were fruit juices (n = 43, 27.6%); sweetened coffee or tea (n = 38. 24.4%); and unflavoured milk (n = 47, 30.1%). The least frequently consumed beverages (< 1 glass/month) were flavoured milk (n = 129, 82.7%); sports drinks (n = 119, 76.3%); and energy drinks (n = 120, 76.9%). The consumption of diet soft drinks was also relatively infrequent (up to 4 glasses per month) among the majority of the participants (n = 109, 69.9%). Relatively few (n = 16, 10.3%) consumed diet soft drinks very frequently (≥ 1 glass/day). The majority (n = 107, 68.6%) of the participants consumed regular soft

drinks infrequently (up to 4 glasses per month), but about (n = 8, 5.1%) consumed regular soft drinks containing added sugar (e.g., Pepsi, Coke, 7-up) every day. Nearly a half of the participants (n = 71, 45.6%) frequently or very frequently consumed alcoholic drinks (1-6/week to ≥ 1/day) whereas less than one third (n = 49, 31.4%) frequently or very frequently consumed regular soft drinks.

Table 2.6 Frequency distributions of the consumption of beverages

Beverage	Category	Frequency	Percent
Fruit juices	< 1/month	14	9.0%
	1-4/month	42	26.9%
	1-6/week	57	36.5%
	≥ 1/day	43	27.6%
Sweetened coffee or tea	< 1/month	49	31.4%
	1-4/month	31	19.9%
	1-6/week	38	24.4%
	≥ 1/day	38	24.4%
Unflavoured milk	< 1/month	32	20.5%
	1-4/month	27	17.3%
	1-6/week	50	32.1%
	≥ 1/day	47	30.1%
Flavoured milk	< 1/month	129	82.7%
	1-4/month	21	13.5%
	1-6/week	5	3.2%
	≥ 1/day	1	0.6%
Sports drinks (e.g., Powerade; Lucozade)	< 1/month	119	76.3%
	1-4/month	25	16.0%
	1-6/week	11	7.1%
	≥ 1/day	1	0.6%
Energy drinks with caffeine	< 1/month	120	76.9%
	1-4/month	24	15.4%
	1-6/week	10	6.4%
	≥ 1/day	2	1.3%
Diet soft drinks (e.g., Diet Pepsi, Coke, 7-up)	< 1/month	73	46.8%
	1-4/month	36	23.1%
	1-6/week	31	19.9%
	≥ 1/day	16	10.3%
Regular soft drinks (e.g., Pepsi, Coke, 7-up)	< 1/month	53	34.0%
	1-4/month	54	34.6%
	1-6/week	41	26.3%
	≥ 1/day	8	5.1%
Alcoholic drinks	< 1/month	37	23.7%
	1-4/month	48	30.8%
	1-6/week	66	42.3%
	≥ 1/day	5	3.2%

2.6.2. Associations between consumption of different types of beverage

Table 2.7 presents a matrix of Cramer's V coefficients, reflecting the extent to which the frequencies of consumption of different types of beverage were associated with each other. The Cramer's V coefficients in Table 2.7 ranged from 0.067 to 0.222, reflecting little or no clinically significant association. There was no statistical evidence to indicate meaningful systematic associations (e.g., that the consumption of one type beverage increased or decreased in proportion to the consumption of another type of beverage). The implications were that the frequency of consumption of each type of beverage was an independent variable, which was not reliant on, or related to, the frequency of consumption of any other type of beverage.

Table 2.7. Matrix of Cramer's V coefficients between the frequencies of consumption of different types of beverage

	Fruit Juices	Sweet-ened coffee /tea	Un-flavour-ed milk	Flavour-ed milk	Alcohol	Sports drinks	Energy drinks	Diet soft drinks
Sweetened coffee / tea	.122							
Unflavour-ed milk	.222	.131						
Flavoured milk	.143	.123	.166					
Alcohol	.160	.113	.168	.112				
Sports drinks	.190	.179	.178	.106	.123			
Energy drinks	.190	.121	.193	.067	.113	.188		
Diet soft drinks	.173	.081	.147	.183	.159	.185	.210	
Regular soft drinks	.188	.151	.157	.143	.167	.222	.146	.161

2.5.3 Associations between consumption of beverages and participant characteristics

Table 2.8 presents a matrix of Cramer's V coefficients to determine the extent to which the frequencies of consumption of different types of beverage were associated with demographic variables (sex and age). The frequency of consumption of most beverages was not associated with the sex of the participants, apart from diet soft drinks, which was weakly associated with sex (Cramer's V = 0.275). There were no clinically significant associations between the consumption of beverages and the age of the participants (Cramer's V = 0.037 to. 0.146).

Table 2.8 Cramer's V coefficients between the frequencies of consumption of beverages with sex and age

Beverage	Demographic characteristics	
	Sex	Age (Years) [a]
Fruit juices	.118	.126
Sweetened coffee or tea	.061	.144
Unflavoured milk	.193	.122
Flavoured milk	.135	.037
Alcohol	.096	.192
Sports drinks	.140	.106
Energy drinks	.173	.146
Diet soft drinks	.275	.056
Regular soft drinks	.125	.092

[a] Two age groups (17-24 and ≥ 25)

The strength and direction of the associations between the categories in the rows and columns of the cross-tabulations were indicated by how far the observed frequencies were away from the expected frequencies (Agresti, 2013). The cross-tabulation in Table 2.9 indicated that among the female participants with low consumption of diet soft drinks (≤ 4/month) the expected frequencies were lower than the observed frequencies, whereas for the males, the expected frequencies were greater than the observed frequencies. Among the females with a higher consumption of diet soft

drinks (≥ 1-6/week) the observed frequencies were less than the expected frequencies (see p.48) but for males the observed frequencies were greater than the expected frequencies. The implications are that a high frequency of consumption of diet soft drinks was lower in females than in males, whereas a low frequency of consumption of diet soft drinks was higher in females than in males.

Table 2.9 Cross-tabulation of observed frequencies of consumption of diet soft drinks vs. sex (expected frequencies in parentheses)

Consumption		Sex		Total
		Female	Male	
Diet soft drinks	< 1/month	43 (41.2)	30 (31.8)	73
	1-4/month	27 (20.3)	9 (15.7)	36
	1-6/week	11 (17.5)	20 (13.5)	31
	≥ 1/day	7 (9.0)	9 (7.0)	16
Total		88	68	156

Table 2.10 presents a matrix of Cramer's V coefficients to determine the extent to which the frequencies of consumption of different types of beverage were associated with the BMI and health characteristics of the respondents.

Table 2.10 Cramer's V coefficients between the frequencies of BMI, health characteristics and consumption of beverages

Beverage	Characteristics		
	BMI	Emotional health [a]	Physical health [b]
Fruit juices	.170	.204	.266
Sweetened coffee or tea	.121	.134	.131
Unflavoured milk	.189	.159	.185
Flavoured milk	.111	.221	.091
Alcohol	.217	.099	.085
Sports drinks	.117	.179	.267
Energy drinks	.064	.139	.251
Diet soft drinks	.165	.048	.201
Regular soft drinks	.112	.259	.074

[a] Two categories (Happy and Unhappy). [b] Two categories (Healthy and Unhealthy).

The emotional and physical health characteristics were originally measured using 5-point scales, but these had to be collapsed to 2-point scales, (to ensure that not more than 50% of the cells in the cross-tabulations contained frequencies < 5). The BMI of the participants was not clinically associated with the consumption of beverages (Cramer's V = 0.067 to 0.217). Emotional health was, however, weakly associated with the consumption of regular soft drinks (Cramer's V = 0.259) and physical health was weakly associated with the consumption of fruit juices (Cramer's V = 0.266) and sports drinks (Cramer's V = 0.267).

The cross-tabulation in Table 2.11 indicated that among the happy participants with a high level of consumption of regular soft drinks (≥ 1-6/week) the observed frequencies were lower than the expected frequencies. Among the unhappy participants with a higher consumption of diet soft drinks (≥ 1-6/week) the observed frequencies were greater than the expected frequencies. The implications are that a high frequency of consumption of regular soft drinks was associated with unhappy participants; whereas a lower frequency of consumption of regular soft drinks was associated with happy participants.

Table 2.11 Cross-tabulation of observed consumption of regular soft drinks and emotional health (expected frequencies in parentheses)

Consumption		Emotional Health		Total
		Unhappy	Happy	
Regular soft drinks	< 1/month	3 (6.8)	50 (46.2)	53
	1-4/month	5 (6.9)	49 (47.1)	54
	1-6/week	9 (5.3)	32 (35.7)	41
	≥ 1/day	3 (1.0)	5 (7.0)	8
Total		20	136	156

The cross-tabulation in Table 2.12 indicated that among the unhealthy participants, the observed frequencies of consumption of fruit juices were consistently higher than expected.

Table 2.12 Cross-tabulation of observed consumption of fruit juices and physical health (expected frequencies in parentheses)

Consumption		Physical Health		Total
		Unhealthy	Healthy	
Fruit Juices	< 1/month	6 (2.3)	8 (11.7)	14
	1-4/month	8 (7.0)	34 (35.0)	42
	1-6/week	4 (3.5)	53 (47.5)	57
	≥ 1/day	8 (7.2)	35 (35.8)	43
Total		26	130	156

Among the healthy participants the observed frequencies were similar to, larger, or lower than, the expected frequencies. The implications are that an unexpectedly high frequency of consumption of fruit juices was associated with unhealthy participants.

The cross-tabulation in Table 2.13 indicated that among the unhealthy participants, the observed frequencies of moderate (1-4/month to 1-6/week) consumption of sports drinks were consistently higher than expected.

Table 2.13 Cross-tabulation of consumption of sports drinks and physical health (expected frequencies in parentheses)

Consumption		Physical Health		Total
		Unhealthy	Healthy	
Sports drinks	< 1/month	14 (19.8)	105 (99.2)	119
	1-4/month	7 (4.2)	18 (20.8)	25
	1-6/week	5 (1.8)	6 (9.2)	11
	≥ 1/day	0 (0.2)	1 (0.8)	1
Total		26	130	156

Among the healthy participants, however, the observed frequencies were consistently lower than expected. The implications are that an unexpectedly high moderate frequency of consumption of sports drinks was associated with unhealthy participants. Infrequent consumption of sports drinks (<1/month) was, however significantly lower than expected among the unhealthy participants, and was greater than expected among the healthy participants.

Table 2.14 presents a matrix of Cramer's V coefficients to determine the extent to which the frequencies of consumption of different types of beverage were associated with the family histories of diabetes, high cholesterol, and heart disease/stroke among the participants. No clinically significant associations were found (Cramer's V = 0.070 to 0.243)

Table 2.14 Cramer's V coefficients between the frequencies of the family history of diseases and the consumption of beverages

Beverage	Family history of diseases		
	Diabetes	High Cholesterol	Heart disease/Stroke
Fruit juices	.185	.141	.124
Sweetened coffee or tea	.190	.185	.154
Unflavoured milk	.204	.081	.230
Flavoured milk	.177	.183	.138
Alcohol	.243	.231	.206
Sports drinks	.241	.103	.127
Energy drinks	.194	.174	.070
Diet soft drinks	.122	.144	.124
Regular soft drinks	.137	.086	.224

Table 2.15 presents a matrix of Cramer's V coefficients to determine the extent to which the frequencies of consumption of different types of beverage were associated with the dietary characteristics of the participants. One clinically significant association, between the consumption of five servings of fruit/vegetables every day and regular soft drinks was found (Cramer's V = 0.292). The expected negative association between lactose intolerant and drinking milk was not found, possibly reflecting the errors in the self-reported data.

Table 2.15 Cramer's V coefficients between the frequencies of the dietary characteristics and the consumption of beverages

Beverage	Dietary characteristics				
	Veget-arian	Lactose intol-erant	Breakfast every day	Five servings of fruits/ vegetables every day	Dietary supple-ments
Fruit juices	.170	.140	.130	.225	.072
Sweetened coffee or tea	.128	.109	.183	.165	.166
Unflavoured milk	.140	.191	.193	.320	.152
Flavoured milk	.149	.134	.185	.148	.166
Alcohol	.149	.145	.215	.166	.109
Sports drinks	.198	.118	.186	.117	.151
Energy drinks	.081	.143	.211	.109	.125
Diet soft drinks	.178	.249	.211	.082	.123
Regular soft drinks	.121	.188	.201	.292	.217

The cross-tabulation in Table 2.16 indicated that among the participants who did not consume five servings of fruit/vegetables every day, the observed frequencies of moderate (1-4/month to 1-6/week) consumption of regular soft drinks were consistently higher than expected. Among the participants who did consume five servings of fruit/vegetables every day, the observed frequencies of moderate (1-4/month to 1-6/week) consumption of regular soft drinks were consistently lower than expected. The implications are that an unexpectedly high moderate frequency of consumption of regular soft drinks was associated with low consumption of fruits/vegetables, and an unexpectedly low consumption of regular soft drinks was associated with a high consumption of fruits/vegetables. Infrequent consumption of regular soft drinks (< 1/month) was, however significantly lower than expected among the participant with lower consumption of fruit/vegetables, participants, and greater than expected among those who consumed five servings of fruit/vegetables every day.

Table 2.16 Cross-tabulation of consumption of regular soft drinks vs. five servings of fruits/vegetables every day

Consumption		Five servings of fruits/ vegetables every day		Total
		No	Yes	
Regular soft drinks	< 1/month	24 (34.3)	29 (18.7)	53
	1-4/month	40 (35.0)	14 (19.0)	54
	1-6/week	31 (26.5)	10 (14.5)	41
	≥ 1/day	6 (5.2)	2 (2.8)	8
Total		101	55	156

Table 2.17 presents a matrix of Cramer's V coefficients to determine the extent to which the frequencies of consumption of different types of beverage were associated with the lifestyle characteristics of the respondents.

Table 2.17 Cramer's V coefficients between the frequencies of the lifestyle characteristics and the consumption of beverages

Beverage	Lifestyle characteristics				
	Smoking	Walking for ten minutes every day	Three strong physical activities /week	Three moderate physical activities /week	Physical activity level [a]
Fruit juices	.083	.233	.158	.107	.184
Sweetened coffee/tea	.208	.128	.126	.063	.194
Unflavoured milk	.043	.159	.180	.084	.129
Flavoured milk	.218	.102	.217	.133	.128
Alcohol	.017	.219	.236	.242	.153
Sports drinks	.159	.065	.177	.126	.054
Energy drinks	.190	.119	.093	.057	.100
Diet soft drinks	.085	.144	.141	.102	.075
Regular soft drinks	.106	.108	.215	.109	.160

[a] Two categories (Inactive and Active)

The physical activity levels were originally measured using a 4-point scale; however, this scale was collapsed to a 2-point scale, (to ensure that not more than 50% of the cells in the cross-tabulations contained frequencies < 5). No clinically significant associations between the frequencies of the lifestyle characteristics and the consumption of beverages could be found (Cramer's V = 0.017 to 0.242).

2.6.4 Associations between diet, lifestyle, and demographic characteristics

Table 2.18 presents a matrix of Cramer's V coefficients to determine the extent to which the diet of the respondents was associated with their demographic characteristics (age and sex). There were no clinically significant associations between the five dietary characteristics and the age of the participants (Cramer's V = 0 .001 to 0.158) or sex (Cramer's V = 0.001 to 0.125) of the participants.

Table 2.18 Cramer's V coefficients between diet and participant characteristics

Demographics	Dietary characteristics				
	Veget-arian	Lactose intolerant	Breakfast every day	Five servings of fruits/ vegetables every day	Dietary supple-ments
Age (Years) [a]	.002	.092	.158	.001	.077
Sex	.107	.125	.106	.001	.052

[a] Two age groups (17-24 and ≥ 25)

Table 2.19 presents a matrix of Cramer's V coefficients to determine the extent to which the frequencies of lifestyle characteristics of the respondents were associated with their demographic characteristics (age and sex). There were no clinically significant associations between the frequencies of the five lifestyle characteristics and the age (Cramer's V = 0.010 to 0.120) or sex (Cramer's V = 0.058 to 0.243) of the participants.

Table 2.19 Cramer's V coefficients between the frequencies of the lifestyle characteristics and the age and sex of the participants

	Lifestyle characteristics				
	Smoking	Walking for ten minutes every day	Three strong physical activities/ week	Three moderate physical activities /week	Physical activity level [b]
Age (Years) [a]	.120	.024	.010	.107	.020
Sex	.058	.066	.243	.218	.139

[a] Two age groups (17-24 and ≥ 25) [b] Two groups (Inactive and Active)

2.7 Discussion

The results of the first survey provided preliminary information to address Research Question 1: What are the dietary and lifestyle risk factors for cardiovascular disease among university students in Edinburgh, Scotland? The risk factors observed among the 156 students included obesity (9.0%); inactive or less active levels of physical inactivity (21.2%); consumption of soft drinks with added or extrinsic sugar every day (5.1%); consumption of alcoholic drinks every day (3.2%); unhealthy diet, implied by not eating five servings of fruits/vegetables every day (64.7%); smoking tobacco (16.7%); as well as a family history of heart disease/stroke (25.6%); high cholesterol (27.6%); and diabetes (46.2%). These findings were consistent with previous surveys concluding that many college and university students have poor compliance with a healthy diet and exhibit relatively low levels of physical activity (Arts et al. 2014; Biswasit, 2015).

The results of the first survey provided only limited information to address Research Question 3: To what extent are the risk factors statistically related to each other? There was no statistical evidence to indicate that the age or sex of the participants were associated with their diet or lifestyle. Only four clinically significant associations (Cramer's V > 0.250) were found between the frequencies of

consumption of different types of beverage by the 156 respondents and their demographic, health, family history of disease, diet, and lifestyle characteristics, as follows. First, the frequency of consumption of diet soft drinks was associated with the sex of the participants (Cramer's V = 0.275). Second, emotional health was associated with the consumption of regular soft drinks (Cramer's V = 0.259). Third, physical health was associated with the consumption of fruit juices (Cramer's V = 0.266) and sports drinks (Cramer's V = 0.267). Fourth, five servings of fruit/vegetables every day was associated with the consumption of regular soft drinks (Cramer's V = 0.292). The expected association between obesity and the consumption of soft drinks containing extrinsic sugars revealed in previous surveys (Bachman et al. 2006; Bawa, 2006; Gill et al. 2006; Malik et al. 2006) was not found.

The interpretation of the Cramer's V coefficients reflected only very weak associations between a few categorical variables linked to CVD risk factors. Consequently, the clinical significance of these results (i.e., their meaningfulness in the context of medical research) were limited. Furthermore, as emphasized by Young (2007, p. 42) in a review entitled "Statistical errors in medical research - a chronic disease", old fashioned methods of analysing associations between categorical variables, developed nearly 100 years ago, such as those based on χ^2 statistics:

> "are to statistics what cupping, bloodletting and leaches are to medicine: of historical interest, on rare occasions still useful, but largely superseded by superior methods. Instead the medical researcher needs to be familiar with their multivariate replacements… I think multivariate methods of analysis should be considered the rule, not the exception."

The use of simple statistics based on χ^2 may partially explain why the categorical responses to the "Diet and Health Screening Questionnaire" did not provide much useful information about the statistical relationships between the CVD risk factors. Furthermore, some of the data collected using this questionnaire could be

inaccurate, because the self-reported responses could be biased. There is considerable evidence to conclude that respondents answering questionnaires concerned with health issues tend to under-report their unhealthy behaviours, such as smoking and drinking alcohol, and over-report their healthy behaviours, such as moderate to high levels of physical activity (Adams et al. 2005; Bener et al. 2003; Brenner & DeLameter, 2014; Hebert et al. 1995; Scagliusi et al. 2003; Choi & Pak, 2005). The tendency of some respondents to exaggerate their socially desirable behaviours, and underestimate their socially undesirable behaviours, is known as social desirability response bias (Holtgreaves, 2004; Mortel, 2007). Socially desirable responding distorts the results of self-report surveys by creating false relationships or obscuring true relationships between variables. Furthermore, self-reported measures of weight and height tend to be inaccurate. Bowering et al. (2012) found that among a sample of 1405 young people (age 16 to 29 years) only 52% accurately self-reported their height, and only 34% accurately self-reported their weight, therefore self-reported estimates of obesity may be misleading. Due to the limitations of the first survey to examine CVD risk factors among students at Heriot-Watt University, associated with the use of simple statistics, and the possibility of response bias, it was essential for the researcher to conduct a second survey, in which (a) most of the data were not measured at the categorical level; (b) the use of self-reported anthropometric measures (e.g., height and weight) were avoided as far as possible; and (c) multivariate statistics were used to analyse the data. Accordingly, the methods and results presented in Chapter 3 were based mainly on the collection and multivariate analysis of data measured at the continuous level.

Chapter Three

Lifestyle, energy/nutrient intake, blood measures and anthropometrics

3.1 Introduction

Despite the possibility of errors due to response bias, the first survey conducted using the "Diet and Health Screening Questionnaire" (see Chapter Two) identified several risk factors for CVD among 156 undergraduate students at Heriot-Watt University. The risk factors included obesity, low physical inactivity, consumption of alcoholic drinks and soft drinks with extrinsic sugar, unhealthy diet, and smoking. The results were consistent with the conclusion that many college/university students have poor compliance with a healthy diet and exhibit relatively low levels of physical activity (Arts et al. 2014; Biswasit, 2015).

These findings justified the implementation of a second survey to collect more useful data on the lifestyle characteristics (dietary constituents, physical activity, consumption of alcohol, smoking tobacco); blood plasma measurements (cholesterol, glucose, and insulin); and anthropometric measures (height, weight, hip and waist) of undergraduate students at Heriot-Watt University. Because continuous level data were collected in the second survey, it was possible to conduct statistical analysis appropriate to this level of data. Multivariate statistics facilitated an exploration of the relationships between the lifestyle characteristics, blood plasma measurements, and the anthropometric measures. The statistical relationships explored using the results of the second survey were underpinned by the model defined by Arts et al. (2014) positing that unhealthy dietary choices among young people are linked to weight gain and obesity, as well as to risk factors such as hypertension, and high levels of blood glucose and LDL-cholesterol.

3.2 Composition of Sample

The sub-sample of 40 students who participated in the second survey during 2014 was not an independent random sample drawn from the target population. This

sample consisted of volunteers who had already completed the "Diet and Health Screening Questionnaire" and who also agreed to continue as participants in the second survey. Because the samples included the same students, the results of the second survey were dependent on the results of the first survey. This implied that inferential statistics assuming independent random sampling, were not justified to directly compare the data collected in first survey and the second study (Hair et al. 2010).

3.3 Anthropometric measurements

Each individual participant was examined in the Sport Academy Centre at Heriot-Watt University between 9:30 am and 12:30 am after an 8 to 12 hour overnight fast. The anthropometric measurements included BMI, body fat, waist and hip circumference, obesity, and blood pressure.

3.3.1 BMI, body fat, and obesity

On arrival, after voiding, the body weight of each participant was measured to the nearest 100 g using a digital balance. The body height of each participant was measured to the nearest 0.5 cm by using a wall-mounted stadiometer. BMI (kg/m^2) was calculated as weight (kg) divided by the height (m) squared. The Body Mass Index (BMI) criteria recommended by the National Obesity Observatory (2009) were used to classify the "fatness" status of the participants. These criteria are appropriate for Caucasian adults aged 18 years and over in the UK, do not change with age, and are the same for both men and women. The BMI level was categorized using a 4-point scale: 1 = Underweight (< 18.5 kg/m^2); 2 = Healthy weight (18.5 to 24.9 kg/m^2); 3 = Overweight (25.0-29.9 kg/m^2); 4 = Obese (≥ 30 kg/m^2). Other measures may provide a better indication of 'fatness' than BMI, because BMI is an indirect and imperfect measurement, not distinguishing between body fat and lean body mass

(Duren et al. 2008; Hu, 2008). Two alternative measures were used in this study. The waist to hip ratio (WHR) was calculated by measuring the waist and the hip at the widest diameter then dividing the waist measurement by the hip measurement). Table 3.1, based on a meta-analysis conducted by Koning et al. (2007) defines the criteria used in this study to classify the WHR of male and female participants into three categories: 1 = low risk; 2 = moderate risk; and 3 = high risk.

Table 3.1 Classification of waist to hip ratio (WHR)

Male	Female	Health risk based on WHR
≤ 0.95	≤ 0.80	Low risk
0.96 to 1.0	0.81 to 0.85	Moderate risk
>1.0	> 0.85	High risk

Source:http://www.bmi-calculator.net/waist-to-hip-ratio-calculator/waist-to-hip-ratio-chart.php

Body fat percentage was measured by bioelectrical impedance analysis (BIA) in a supine position. Single frequency bioelectrical impedance at 50 kHz was passed between surface electrodes placed on the hand and foot of each student to determine the fat-free mass and total body water. A published BIA equation validated against a reference method was used to estimate body fat percentage (Kyle et al. 2004). Table 3.2 shows how the percentage body fat was assessed according to sex and age (between 20 to 39 years). The categories used to classify the percentage body fat in male and female students was 1 = low, 2 = healthy, 3 = increased, and 4 = high; as defined in Table 3.2 (Royal College of Nurses, 2009).

Table 3.2 Classification of body fat percentage

Assessment	Body fat (%)	
	Female	Female
1 = Low	< 21	< 8
2 = Healthy	21-33	8-20

3 = Increased	33-39	20-25
4 = High	> 39	> 25

3.3.2 Hypertension

The systolic and diastolic blood pressure of each student was measured using an automatic cuff based on the principle of oscillometry. The automatic cuff is an air-filled balloon which was wrapped in a non-expanding fabric around the arm of the student. The cuff was inflated to well above systolic blood pressure, until the brachial artery collapsed, and then automatically vented through the bleed valve. As the pressure in the cuff slowly decreased, the blood began to flow through the artery. The pressure in the cuff when blood first began to pass through was estimated electronically to provide the systolic blood pressure. The pressure in the cuff when blood began to pass continuously was then estimated to provide the diastolic blood pressure. The digital readout of the systolic and diastolic blood pressures was recorded. The oscillometric method of measuring blood pressure has been validated; however, some researchers have questioned its validity in screening for hypertension because it may sometimes provide pressure readings that are too high (Babbs, 2012).

The chart in Figure 3.1, applicable for adults over 18 years old in the UK, was used to classify the blood pressures of the participants in this study, in units of mm Hg. The 4-point scale was as follows: 1 (90/50 to 90/40) = Low; 2 (90/80 to 120/80 = Ideal; 3 (120/80 to 140/90) = Pre-high blood pressure; 4 (140/90 to 190/100) = high blood pressure (hypertension).

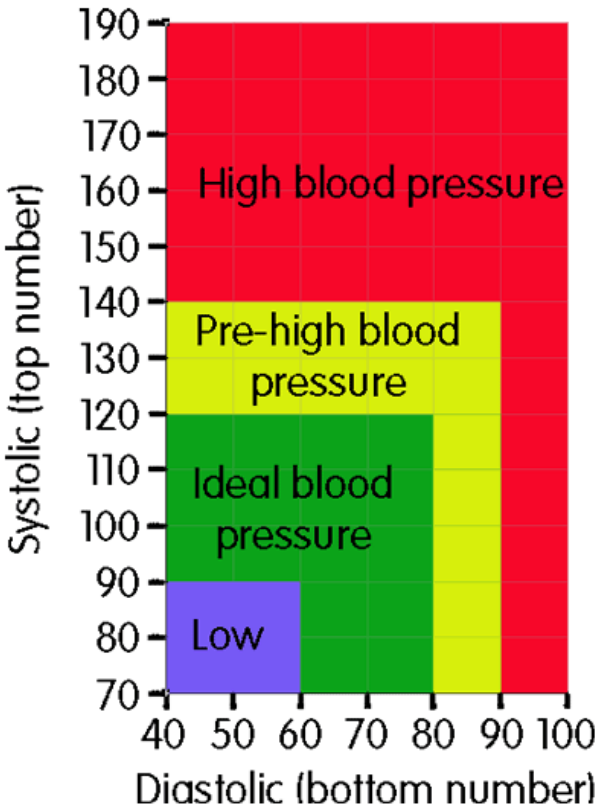

Figure 3.1 Classification of blood pressure

Source:
http://www.bloodpressureuk.org/BloodPressureandyou/Thebasics/Bloodpressurechart

3.4 Daily energy and nutrient intake

Daily energy and nutrient intakes were determined by questionnaire. The participants were asked not to change their usual eating habits during the three days that they recorded all of the food items that they consumed. The three-day food records for each student were checked to ensure completeness and comprehensibility. The food item data were then entered into a nutritional analysis programme (WinDiets Research software, available at http://www2.rgu.ac.uk/windiets/wresearch.htm) to convert the amount of eaten food to energy and nutrient items, as listed in Table 3.3

Table 3.3 Energy and nutrients computed by WinDiets software

Alcohol	g
Alpha tocopherol equivalents	mg
Ascorbic acid (Vitamin C)	mg
Beta carotene equivalents	µg
Biotin	µg
Calcium	g
Carbohydrate	g
Chloride	mg
Cholesterol	mg
Copper	mg
Dietary Fibre	g
Energy	kJ
Energy value	kcal
Fat	g
Iodine	µg
Iron	mg
Magnesium	mg
Manganese	mg
Monounsaturated fatty acids	g
Nicotinic acid equivalent	mg
Non-milk extrinsic sugars	g
Non-starch polysaccharides	g
Pantothenic acid	mg
Phosphorus	mg
Polyunsaturated fatty acids	g
Potassium	mg
Protein	g
Retinol	µg
Retinol equivalents	µg
Riboflavin (Vitamin B_2)	mg
Saturated fatty acid	g
Selenium	µg
Sodium	mg
Starch	g
Thiamine (Vitamin B_1)	mg
Total folate	µg
Total sugars	g
Vitamin B_{12}	mg
Vitamin B_6	mg
Vitamin D	µg
Water	g
Zinc	mg

3.4.1 Evaluation of energy and nutrient intake

Two criteria could be used to evaluate the dietary constituents of the participants. The first was the Dietary Reference Values (DRVs) for dietary items published by the Department of Health (1991) in the UK, listed in Table 3.4

Table 3.4 Dietary reference values for adult males and females in UK

Item	DRV	Units/day
Calcium	700	mg
Folic acid	200	µg
Iron	8.7	mg
Protein	55.5	g
Riboflavin	1.3	mg
Sodium	1600	mg
Thiamine	1	mg
Non-Starch Polysaccharide	18	g
Polyunsaturated Fat	18.6	g
Vitamin C	40	mg
Niacin	17	mg
Phosphorus	550	mg
Vitamin B_{12}	1.5	µg
Saturated Fat	31.5	g
Energy value (kcal)	2531	kcal
Energy (MJ)	10.6	MJ
Fat	100	g
Magnesium	300	mg
Non-Milk Extrinsic Sugars	73	g
Potassium	3500	mg
Vitamin A	700	µg

The DRVs are intended to serve only as guidance for good nutrition. They are not recommended daily intakes for the UK population, but are statistical concepts, provided to aid the interpretation of dietary information for both groups and individuals. The DRVs are based on an assessment of average estimates of 22 nutrients for the UK population as a whole, and, with the exception of energy, are not stratified by gender or age group. The second criterion was devised in the USA, in which equivalent values to the DRVs were called Dietary Reference Intakes (DRIs). The DRIs include 33 nutrients, classified according to gender and age (National

Academy of Sciences, Institute of Medicine, Food and Nutrition Board, 2010). The DRIs for men and women of age 19 to 30 years are listed in Table 3.5

Table 3.5 Dietary reference intakes (DRIs) for males and females age 19-30 in USA

Item	DRI	Units/day
Carbohydrate	130	g
Chloride	2300	mg
Copper	0.9	mg
Dietary Fibre	38	g
Energy value	2000	kcal
Energy	10.6	MJ
Fat	100	g
Folic acid	320	µg
Iodine	150	µg
Iron	8	mg
Magnesium	400	mg
Manganese	2.3	mg
Niacin	12	mg
Non-Starch Polysaccharide	18	g
Non-Milk Extrinsic Sugars	73	g
Phosphorus	700	mg
Polyunsaturated Fat	18.6	g
Potassium	4700	mg
Protein	56	g
Retinol	35	µg
Riboflavin	1.1	mg
Saturated Fat	3	g
Selenium	55	µg
Sodium	1500	mg
Thiamine	1	mg
Vitamin A	625	µg
Vitamin B_{12}	2	µg
Vitamin B_6	1.1	mg
Vitamin C	75	mg
Vitamin D	10	µg
Vitamin E	12	mg
Water	1850	g
Zinc	11	mg

Because the DRI's are more comprehensive that the DRVs, and are based on more recent research, the DRI's were applied in this study. The DRI value of each item in Table 3.5 was subtracted from the measured value, resulting in a positive number (if the measured value was greater than the DRI) or negative numbers (if the

measured value was less than the DRI). To classify the students into two groups, depending on whether their dietary constituents were above or below the DIR values, the positive values were coded as 1 and negative values were coded as 0.

3.5 Consumption of alcohol

The consumption of alcohol by each participant was measured in g/day; however, there is no recommended alcohol intake in g per day. The number of units in an alcoholic drink is based on the size of the drink as well as its alcohol strength, where 1 unit = strength of alcohol by volume (%) x volume (ml) / 1000, and 1 unit is equivalent to 8 g of alcohol. The National Health Service (2015) guidelines for the UK population state that increased risk of damage to health may be caused by regular drinking of 3-4 units of alcohol (24 to 32 g) per day for men, and regular drinking of more than 2-3 units (16 to 24 g) per day for women. In this study the alcohol use of each participant was determined by whether or not his or her daily intake of alcohol was above or below these guidelines.

3.6 Blood plasma measurements

All participants were asked to avoid consuming alcohol and fatty evening meals and not to indulge in active physical activity two days before the examination, because this could affect their blood pressure and blood glucose levels. Blood samples were collected for the measurement of the average plasma concentrations of lipoproteins, insulin, and blood glucose level on two occasions, at baseline, and six to twelve months after baseline. The blood samples were centrifuged for 15 minutes at 3000×g at 10° C to separate the plasma. The plasma was stored at -70□ before the measurements were made.

3.6.1 Lipoproteins

Lipoproteins are particles in the blood that carry cholesterol, and include (a) LDL-cholesterol (low-density lipoprotein cholesterol, also colloquially called "bad" cholesterol) and (b) HDL-cholesterol (high-density lipoprotein cholesterol, also called "good" cholesterol). Blood samples (100 ml) were collected from each student, and centrifuged for 15 minutes at 3000×g at 10° C to separate the plasma. The plasma was stored at -70□ before the measurements were made. HDL and LDL were measured in the blood samples using a Cholesterol Assay Kit (Cell Biolabs, 2012). This kit included reagents for separating and quantifying HDL-cholesterol and LDL-cholesterol in a 96-well micro plate format. The colorimetric assay was based on an enzyme driven reaction. Cholesterol esters were hydrolysed via cholesterol esterase into cholesterol, which was then oxidized by cholesterol oxidase into the ketone cholest-4-en-3one and hydrogen peroxide. The hydrogen peroxide was detected with a fluorescence probe. Samples were compared to known standard concentrations of cholesterol using a calibration curve. The samples and standards were incubated for 45 minutes and then readings were taken with a standard 96-well fluorometric plate reader.

The criteria recommended by the executive summary of the third report of the National Cholesterol Education Programme (2001) were used to classify the LDL-Cholesterol levels of the participants in this study. The 5-point scale was: 1 = Optimal (< 100 mg/dL); 2 = Near/Above Optimal (100-129 mg/dL); 3 = Borderline High (130-159 mg/dL); 4 = High (160-189 mg/dL); 5 = Very High (> 190 mg/dL). The participants were also divided into whether their HDL levels were 1 = Optimal (> 60 mg/dL); 2 = Near Optimal (40-59 mg/l) or 3 = Sub-optimal (< 40 mg/dL). The

relative risk of CVD was indicated by the ratio of LDL to HDL as indicated in Table 3.6 where 1 = very low; 2 = average, 3 = moderate, 4 = high.

Table 3.6 Classification of ratio of LDL/HDL

Risk	Ratio of LDL/HDL	
	Men	Women
Very low	1	1.5
Average	3.6	3.2
Moderate	6.3	5.0
High	8	6.1

Source: http://www.exrx.net/Testing/LDL%26HDL.html

3.6.2 Insulin

Plasma insulin concentrations were measure by Enzyme-Linked-Immune-Sorbent-Assay (ELISA) according to the manufacturers' instructions (Mercodia, 2010) ELISA is a two-site enzyme immunoassay for the quantification of human insulin in serum or plasma based on the sandwich technique. Two monoclonal antibodies are directed against separate antigenic determinants on the insulin molecule. Monoclonal antibodies are mono-specific toward a single part of an antigen molecule to which an antibody attaches itself allowing detection and quantitation of small differences in antigen. Insulin reacts with the anti-insulin antibodies bound to microtitration wells and peroxidase-conjugated anti-insulin antibodies in the solution.

Insulin was measured in "micro-units per millilitre" (mU/ml). The lowest detection limit is 1 mU/ml. Because of the high level of variability within and between individuals, there is not much agreement in the literature as to the ideal level of insulin (Spero, 2013). The average insulin level is about 8.6 mU/ml. The participants were classified into two groups, according to whether their insulin level was either ≤ 8.6 mU/ml or > 8.6 mU/ml.

3.6.3 Glucose

An Oral Glucose Tolerance Test (OGTT) was conducted for each participant. The OGTT test was used because it is currently the gold standard for the diagnosis of diabetes. The shape of the glucose response curve during an OGTT identifies physiologically different groups of individuals with differences in insulin secretion. However, the sensitivity of the test to incorrect preparation or administration and the high variability of blood glucose levels within each individual makes this test less than perfectly reliable. The recommended methods of preparation for and administration of the OGTT were carefully implemented to ensure that test results were accurate (Phillips, 2012).

At 9:30 after an 8 to 12-h overnight fast, each participant was asked to lay on a bed in a comfortable position. Baseline, fasting blood samples were obtained using a finger stick. Then the participant was asked to consume a glucose drink (75g of glucose dissolved in 200 ml of water) and a further blood sample was taken after 120 minutes using a finger stick. During the test no carbohydrate was consumed and the student remained seated throughout the two hours of the test.

Glucose tolerance was measured using the HemoCue® Glucose 201 System. This is a hand-held and battery-operated system ideal for mobile settings using microcuvette technology. The before and after meal blood glucose concentrations were provided by electronic meter readings. This method is not necessarily very precise. The meter readings of blood glucose concentrations are generally within 20% of their true values. More precise blood glucose measurements may be performed in a medical laboratory, using hexokinase, glucose oxidase, or glucose dehydrogenase enzyme systems (Olansky & Kennedy, 2010).

Normal blood glucose concentration should be about 4 mmol/L. Shortly after a meal the blood glucose level may rise temporarily up to 7.8 mmol/L. The participants

were classified according to the International Diabetes Federation (2007) target levels for average before meals (pre-prandial) and after meals (post prandial) blood sugar levels measured with two separate tests, as shown in Table 3.7.

Table 3.7 Classification of blood glucose levels

Type	Blood glucose (mmol/L)	
	Before meals	2 hours after meals
Non-diabetic	4.09-5.9	< 7.8
Type II diabetes	4.0-7.0	< 8.5
Type I diabetes	4.0-7.0	< 9.0

Source: http://www.diabetes.co.uk/diabetes_care/blood-sugar-level-ranges.html

3.7 Smoking tobacco

There is only one generally recommended daily exposure to avoid the health risks of tobacco smoke and that is zero. Not smoking is an important part of a healthy lifestyle, including maintaining a healthy weight, consuming a healthy diet, and being physically active. The participants in this study were classified according to whether they self-reported 1 = “Yes” or 0 = “No” to the question about smoking.

3.8 Physical activity

The physical activity of each participant was classified using a simple 3-point self-reported scale: 1 = less active person, 2 = moderately active person, 3 = very active person.

3.9 Multivariate analysis of CVD risk factors

Multivariate techniques were used to classify the participants according to multiple risk factors. These techniques included cluster analysis and principal component analysis (Hair et al. 2010). The use of multivariate statistics in medical research is based on the principles that (a) few, if any clinical, physiological or pathological factors in the human body operate in isolation, because everything is linked together into a complex network of interacting factors which operate as a

continuum; and (b) the similarities between groups of participants identified by linking together a multiplicity of factors, are more likely to be clinically significant and realistic than the differences between groups of subjects identified by evaluating the effects of individual factors considered in isolation.

3.9.1 Cluster analysis

Cluster analysis is a method of classification that organizes a large number of individuals into smaller groups or clusters. The closer the proximity of the clusters, then the more similar are the characteristics of the individuals in each cluster. The farther apart are the clusters, then the more dissimilar are the individuals in each cluster (Everitt et al. 2001). The justification for using cluster analysis in the current study was that, for the purpose of medical and healthcare research, this multivariate technique offers a powerful approach to facilitate the extraction of relatively homogenous sub-groups of participants from data containing considerable heterogeneity in terms of clinical, anatomical, physiological, pathological, or other human measurements. Cluster analysis has been used for this purpose for over twenty-five years (McLachlan, 1992; Clatworthy et al. 2005; Haldar et al. 2008; Liao et al. 2016). Cluster analysis has recently been used to classify different groups of patients diagnosed with CVD in order to guide clinical management. Different clusters were significantly associated with a wide range of different clinical outcomes (Ahmad et al. 2016; Hertzog et al. 2010).

Cluster analysis was conducted in this study using MINITAB version 17.1 software to classify the 40 participants into separate groups with respect to their CVD risk factors, specifically their anthropometric measurements, blood plasma measurements, and dietary constituent items. Because cluster analysis is not an inferential statistical method, there were no test statistics or p-values. In the tradition

of exploratory data analysis, the interpretation of the results of cluster analysis was inductive, to generate hypotheses rather than deductive, to test hypotheses (Hair et al. 2010). The closer the proximity of the clusters, then the more similar were the CVD risk factors of the participants. The farther apart were the clusters, then the more the CVD risk factors were dissimilar.

Several methods were possible to link the clusters, including Single, Average, McQuitty, and Ward. Several distance measures were also possible to separate the clusters, including Euclidean and Pearson (Aldenderfer & Blashfield, 1984). Ward's linkage method was chosen, because this method, based on the analysis of sums of squares, is reported to be the most efficient method of discriminating between different groups of individuals. Squared Euclidean distance was chosen, because this distance measure is commonly used in combination with Ward's linkage to ensure that the participants were as widely separated as possible on different branches of each cluster (Everitt et al. 2001).

A tree structure or dendrogram was drawn, consisting of ranked groups of participants, in order to interpret the results of the cluster analysis. The dendrogram consisted of distinct cluster groups, each of which contained individuals sharing common risk factors. Each cluster group was separated by major dichotomous branches, identified visually as black lines. The black lines represented the lowest levels of similarity between the CVD risk factors. At the end of each major dichotomous branch, the participants were separated on individual dichotomous branches, to create distinct cluster groups. Minitab identified each distinct cluster group using a different colour. Finally, a profile of each cluster group was created, to compare the CVD risk factors presented by the participants in each cluster.

3.9.2 Principal component analysis

Principal component analysis (PCA) is a method of ordination, meaning that it identifies relationships and patterns among multivariate data. PCA reduces multidimensional data into a simpler and more understandable form, by transforming the data into a smaller number of dimensions called principal components. In contrast to cluster analysis, which assigns individuals into separate groups, PCA detects continuous variation between individuals (Joliffe, 2002).

The justification for using PCA in the current study was that, for the purpose of medical and healthcare research, this multivariate technique offers a powerful approach to facilitate the reduction of multidimensional data into a smaller number of dimensions. PCA has been applied for this purpose for over forty years (Barber et al. 1975). With respect to research on CVD, PCA has been applied to large sets of clinical data collected from patients diagnosed with CVD in order to extract a scale of risk factors for metabolic syndrome (Argawal et al. 2012). Bhuvaneswari (2013) developed a medical diagnosis system to predict the risk of CVD based on the use of PCA. A multidimensional dataset based on measurements of CVD symptoms with 13 attributes was reduced to 7 principal components using PCA.

In the current study PCA was conducted to extract the principal components from the anthropometric measurements, blood plasma measurements, and dietary constituent items using MINITAB version 17.1 software. The first component accounted for the highest variance in the data. The next component accounted for less variance, the third component accounted for the least variance. The PCA scores for each participant were plotted in three-dimensional (3-D) scatter plots were interpreted inductively to generate hypotheses. The orientation of PCA scores in the 3-D scatter plot indicated the similarities and differences between the participants. Closely aggregated groups of PCA scores were assumed to represent closely related

participants (similar to a cluster group in cluster analysis), whereas points spaced widely apart were assumed to represent distantly related or unrelated participants.

The grouping of the participants derived by PCA was compared with the grouping of the participants obtained using cluster analysis. If the results of both cluster analysis and PCA based on completely different statistical approaches were comparable, then the grouping of the participants could be considered to be stable and reliable (Shaw, 2003).

3.9.3 Structural equation modelling

Several methods could be used to explore the statistical relationships between the risk factors collected in this study, including correlation, regression, and structural equation modelling (SEM). SEM was chosen because it is a modern second-generation technique that offers many advantages over older first-generation techniques such as correlation and regression analysis devised nearly 100 years ago (Alavifar, et al. 2012). SEM has previously had limited use in medical and healthcare research because it is a modern technique. However, the applications of SEM in medical and healthcare research have expanded rapidly in the last decade (Beran & Violao, 2010; Berglund et al. 2012; Christ et al. 2014; Lee, 2008; Rabe-Hesketh & Skrondal, 2008) justifying the use of SEM for the purpose of the current study. In the context of research on CVD, Mi et al. (2011) used SEM to formulate a model that explored the statistical relationships between gene–environment interactions in coronary heart disease patients classified by genotype (SERPINE1 and APOE); age, sex, BMI, smoking cigarettes and drinking alcohol. These environmental factors were correlated with the patient's levels of BG (different genetic carriers); hypertension, total cholesterol (TC), and HDL-cholesterol, which were ultimately related to coronary heart disease. A diagram of this model is depicted in Figure 3.2.

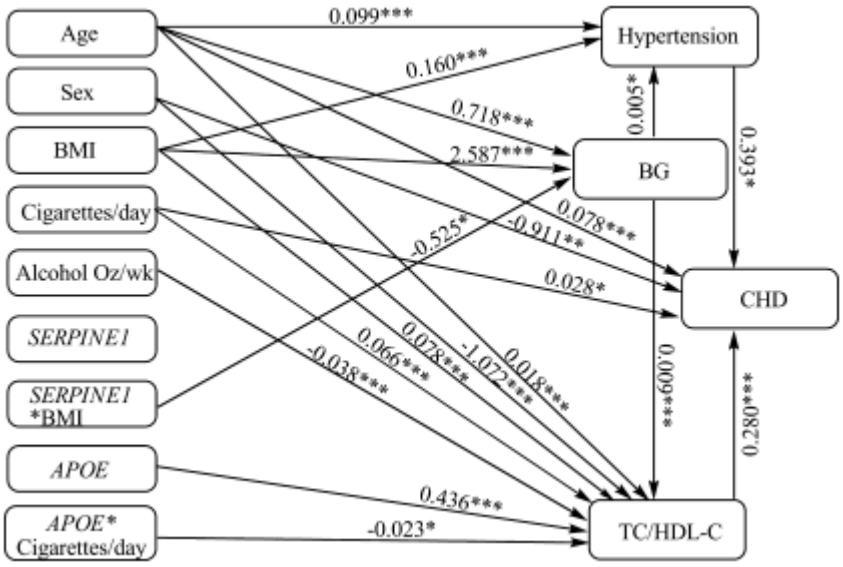

Figure 3.2 SEM model of relationships between gene–environment interactions in CHD patients (Mi et al. 2011).

Figure 3.2 displays the essential features of a structural equation model, including the measured variables (represented by rectangular or oval symbols); the relationships between the variables (symbolized by arrows); and the path coefficients (the numbers next to the arrows). A path coefficient is a standardized regression coefficient estimating the strength and direction of the relationship between an independent variable and a dependent variable. The model in Figure 3.2 explains that increasing age, cigarette smoking, being male, hypertension, and high TC/HDL-c are major risk factors for CHD. The CHD risk factors interact with the genetic factors (SERPINE1, APOE, and BG).

There are two approaches to SEM. The first is called covariance-based SEM (CB-SEM), using software such as AMOS (Hair et al. 2010). The purpose of CB-SEM is to create an empirical statistical model that is a good fit to the covariance matrix. The second approach is SEM based on partial least squares (PLS-SEM) using

software such SmartPLS, (Wong, 2013). PLS-SEM does not attempt to reproduce the covariance matrix, but attempts to optimize the explained variance (Hair et al. 2010).

PLS-SEM was chosen for the current study because it can operate effectively with a lower quality and quantity of data than CB-SEM (Hair et al. 2010). PLS-SEM is a non-parametric method, meaning that it can operate with all types of categorical and interval scaled variables, and it is not based on the assumption that the variables are normally distributed (Hair et al. 2011; 2014). Rigdon (2012) praised PLS-SEM because of its simplicity compared to alternative complex modelling methods such as CB-SEM. Furthermore, PLS-SEM has a high level of statistical power to compute an accurate model, even if the sample size is small (as low as 30); whereas CB –SEM has less power, and requires a very large sample size (usually at least 200). Consequently, the sample size of 40 participants used in the current research was adequate for PLS-SEM.

SmartPLS was the software used in this study to conduct PLS-SEM using the protocols described by Wong (2013). The path diagram drawn using the graphic user interface of SmartPLS is illustrated in Figure 3.3 to examine the relationships between the CVD risk factors measured in the current study. The blue oval symbols (Blood measurements, Unhealthy diet and lifestyle, Healthy diet and lifestyle, and Anthropometric measurements) are the latent variables that were not directly measured by the researcher, but were operationalized by aggregating clusters of measurements called formative indicators, defined by the yellow rectangular symbols. SmartPLS used composite factor analysis to formulate the latent variables as a linear combination of the indicators. The path coefficients (denoted β_1, β_2, and β_3) estimated the strengths and directions of the partial correlations (i.e., the correlations between pairs of latent variables after the effects of the correlations with other latent variables

had been excluded, or partialled out). Consequently, the path coefficients were not equivalent to bivariate correlation coefficients.

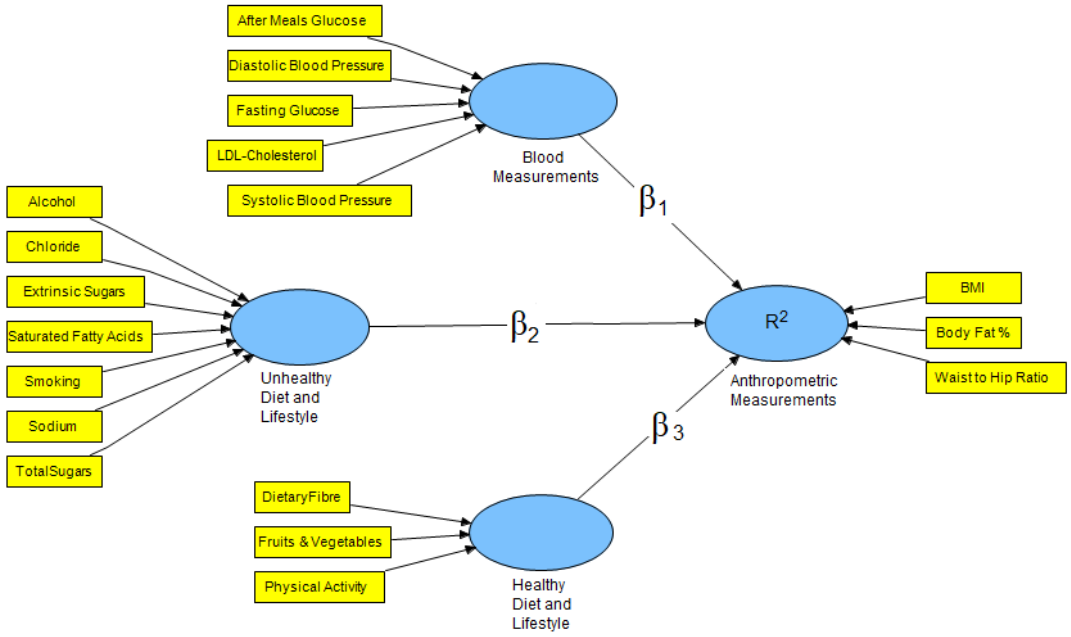

Figure 3.3 Structural equation model drawn by SmartPLS

Note: Ovals = latent variables; Rectangles = indicators; β_1, β_2 and β_3 = path coefficients

The path diagram included two types of latent variable, called exogenous and endogenous. The exogenous latent variables, representing the CVD risk factors did not have any other latent variables with arrows directed into them and were equivalent to the independent variables in a regression model. The endogenous latent variable, with three arrows directed into it, representing the anthropometric measurements, was equivalent to the dependent variable in a regression model. The PLS-SEM analysis involved the computation and evaluation of path coefficients (β) and effect sizes (R^2). The standardized path coefficients (β1, β2, and β3 in Figure 3.3) measured how much of the multidimensional variance in the data was explained by the associations between the latent variables. The magnitude of each β coefficient (ranging from -1 through 0 to +1) indicated the direction (positive or negative) and strength of the

association between the endogenous variable and each exogenous variable. The R^2 value measured the effect size, reflecting the proportion of the multidimensional variance explained in the endogenous variable by the three endogenous variables directed into it. Although the path diagram may appear to represent causal relationships, it was not possible to identify causal relationships, because the data were collected in a cross-sectional

3.10 Results

The results based on the data collected in the second survey are presented systematically in three sections: 3.11 Characteristics of participants; 3.12 Univariate analysis of risk factors; 3.13 Multivariate analysis of risk factors.

3.11 Characteristics of participants

The distributions of the frequencies of the age and sex of the participants are cross-tabulated in Table 3.8. The sample consisted of approximately equal numbers of female (19, 47.5%) and male (n = 21, 52.5%) students. They ranged in age from 18 to 24 years old. One half of the sample (n = 20, 50.0%) were between 18 and 20 years old and the other half (n = 20, 50.0%) were between 21 and 24 years old. The sex and age distributions of the sample were considered to be representative of the student population.

Table 3.8 Frequency distributions of the age and sex of the participants

Age (Years)	Sex		Total
	Female	Male	
18	4 10.0%	2 5.0%	6 15.0%
19	2 5.0%	4 10.0%	6 15.0%
20	1 2.5%	7 17.5%	8 20.0%
21	3 7.5%	0 0.0%	3 7.5%
22	3	1	4

	7.5%	2.5%	10.0%
24	2	4	6
	5.0%	10.0%	15.0%
Total	19	21	40
	47.5%	52.5%	100.0%

3.12 Univariate analysis

Univariate analysis means a descriptive analysis of individual variables, without considering their relationships or interactions with other variables (Hair et al. 2010). This section presents the results of the univariate classification of the participants by CVD risk factors (anthropometric measurements, smoking tobacco, physical activity, diet, consumption of alcohol, hypertension, cholesterol and diabetes). In this analysis, each variable is only considered a single risk factor, without reference to the others.

3.12.1 Anthropometric measurements

The anthropometric measurements of the 40 participants are summarized in Table 3.9. Based on the mean values, the sample generally had: (a) a healthy BMI (23.92 kg/m^2); (b) a low risk waist to hip ratio (0.76); (c) a low body fat (19.68%); The wide range of measurements, however, indicated that the sample included participants who ranged from underweight to obese.

Table 3.9 Summary of anthropometric measurements

Measurement	Mean	SD	Min	Max	Range
BMI (kg/m^2)	23.92	2.55	18.20	31.0	12.8
Waist to hip ratio	0.76	0.07	0.64	1.00	0.36
Body fat (%)	19.68	7.05	5.90	33.70	27.80

The frequency distribution of BMI among the 40 participants was classified according to the National Obesity Observatory (2009) scheme using a 4-point scale: 1

= Underweight (< 18.5 kg/m^2); 2 = Healthy weight (18.5 to 24.9 kg/m^2); 3 = Overweight (25.0-29.9 kg/m^2); 4 = Obese (≥ 30 kg/m^2). Figure 3.4 illustrates the frequency distribution histogram of BMI. The majority of the participants (n = 24, 60.0%) had a healthy weight, but over one third r (n = 13, 32.5%) were overweight. The sample also included two participants (n = 2, 5%) who were classified as obese.

The waist to hip ratio (WHR) ranged from 0.65 to 0.76 in the female students and 0.65 to 1.00 in the male students. One male student had a moderate health risk due to a high WHR (1.00). The body fat percentage ranged from 21.1% to 33.7% in female students, and 5.9% to 30.1% in male students. Based on body fat > 25% a substantial proportion (n = 7, 17.5%) of the male students were at risk due to increased body fat. Based on body fat > 39% none of the female students were at risk.

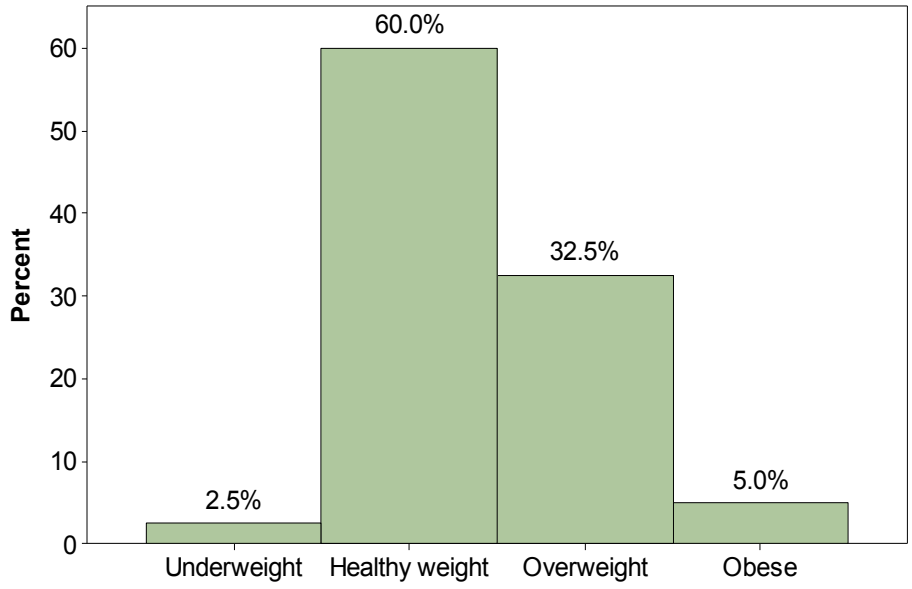

Figure 3.4 Frequency distribution of BMI categories

3.12.2 Physical activity

Figure 3.5 depicts the frequency distribution histogram for the physical activity of the participants. Less than one quarter (n = 9, 22.5%) reported that they were less active, implying a greater risk of CVD. Over half (n = 22, 55.0%) described themselves as moderately active, and less than one quarter (n = 9, 22.5% described themselves very active.

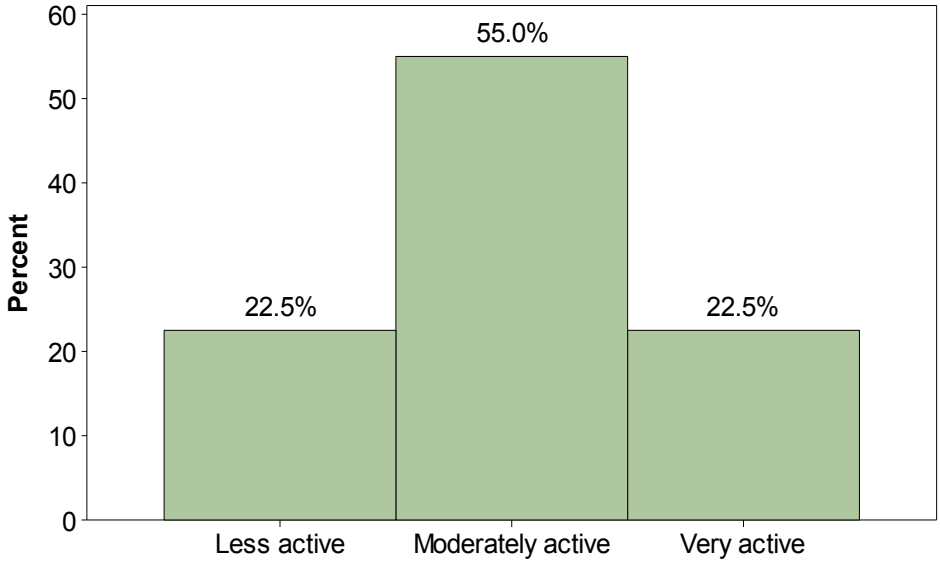

Figure 3.5 Frequency distribution of physical activity categories

3.12.3 Diet

Table 3.10 shows that the majority of the participants (n = 32, 88.0%) admitted that they did not eat five servings of fruit/vegetables every day.

Table 3.10 Consumption of fruit/vegetables

Characteristic	Category	n	%
Eat five servings of fruit/vegetables every day	No	32	88.0
	Yes	8	20.0

Table 3.11 presents the descriptive statistics for the mass of different dietary items consumed per day, based on the three-day food records of the 40 participants. Some of the standard deviations (SD) were greater than the means (e.g., for alcohol

and dietary fibre) reflecting the very wide range of consumption relative to the average for these items. The standardized differences between the DRI values and the mean mass of each item consumed per day were computed to indicate how far away (positive or negative) the mass of each consumed item was from the DRI value. The standardized differences permitted comparison of the DRI vs. the consumed items in units of SD, even though the units of measurement of the items were not the same, and the SDs, as a proportion of the means, were highly variable. The most negative difference (indicating that, on average, the students consumed more than one SD less than the DRI values) were for Selenium (-1.9); Dietary Fibre (-1.7); and Water (-1.5). Low intake of dietary fibre may play a role in the apparent protective effect of dietary fibre on the risk of CVD (Bazzano et al. 2003). The most positive difference (indicating that, on average, the students consumed more than one SD more than the DRI values) were for Sodium (1.1); Phosphorus (1.3); Protein (1.4); Saturated fatty acid (1.7); and Energy (2.7). These results reflect an unhealthy diet, defined as the consumption of modern convenience foods, rich in saturated fatty acid and salt (Crawford, 2013).

Table 3.11 Summary of items consumed compared to Dietary Reference Intake (DRI) values

Item	Units	DRI	Mean	SD	(Mean -DRI) /SD
Alcohol	g		3.88	8.84	
Beta carotene equivalents	µg		1939.1	2692.2	
Biotin	µg		44.2	49.1	
Calcium	mg		977.7	532.6	
Cholesterol	g		345.5	236.5	
Mono-unsaturated fatty acids	g		28.7	12.6	
Nicotinic acid equivalent	mg		42.8	17.5	
Pantothenic acid	mg		6.3	4.2	
Starch	g		118.2	60.2	
Total sugars	g		87.1	44.6	
Selenium	µg	55	30.3	12.9	-1.9
Dietary Fibre	g	38	8.9	16.9	-1.7

Water	g	1850	1138.7	489.3	-1.5
Potassium	mg	4700	3144.2	1714.8	-0.9
Poly-unsaturated fatty acids	g	18.6	13.7	6.3	-0.8
Folic acid	µg	320	245.9	130.0	-0.6
Non-milk extrinsic sugars	g	73	58.3	32.7	-0.4
Fat	g	100	89.0	32.7	-0.3
Non-starch polysaccharides	g	18	13.2	15.6	-0.3
Iodine	µg	150	127.1	92.7	-0.2
Magnesium	mg	400	362.8	243.9	-0.2
Vitamin D	µg	10	9.1	4.8	-0.2
Vitamin C	mg	75	77.5	53.4	0.0
Energy value	kcal	2000	2057.2	658.1	0.1
Vitamin A	µg	35	36.5	28.9	0.1
Zinc	mg	11	12.4	7.8	0.2
Copper	mg	0.9	1.9	1.9	0.5
Manganese	mg	2.3	3.6	2.5	0.5
Chloride	mg	2300	4509.7	3059.9	0.7
Iron	mg	8	17.6	12.9	0.7
Vitamin B_1	mg	1	1.8	1.1	0.7
Vitamin B_2	mg	1.1	2.0	1.0	0.9
Vitamin B_{12}	mg	2	5.3	3.7	0.9
Vitamin B_6	mg	1.1	2.3	1.3	0.9
Carbohydrate	g	130	231.4	95.3	1.1
Sodium	mg	1500	3502.6	1883.1	1.1
Phosphorus	mg	700	1621.3	733.5	1.3
Protein	g	56	96.0	28.0	1.4
Saturated fatty acid	g	3	60.1	34.5	1.7
Energy	MJ	10.6	86.4	27.7	2.7

3.12.4 Hypertension

Table 3.12 presents a summary of blood pressure measurements, indicating that, on average, the students had ideal blood pressure (90/80 to 120/80). However, the wide range in blood pressure measurements indicated that some students did not have ideal blood pressure.

Table 3.12 Summary of blood pressure measurements

Blood pressure	Mean	SD	Min	Max	Range
Systolic (mm Hg)	112.41	9.62	96.00	142.50	46.5
Diastolic (mm Hg)	76.09	8.64	56.00	94.50	38.5

The frequency distribution of the blood pressures of the participants was classified using a 4-point scale: 1 = Low; 2 = Ideal; 3 = Pre-high blood pressure; 4 = high blood pressure (hypertension). The frequency distribution histogram is illustrated in Figure 3.6.

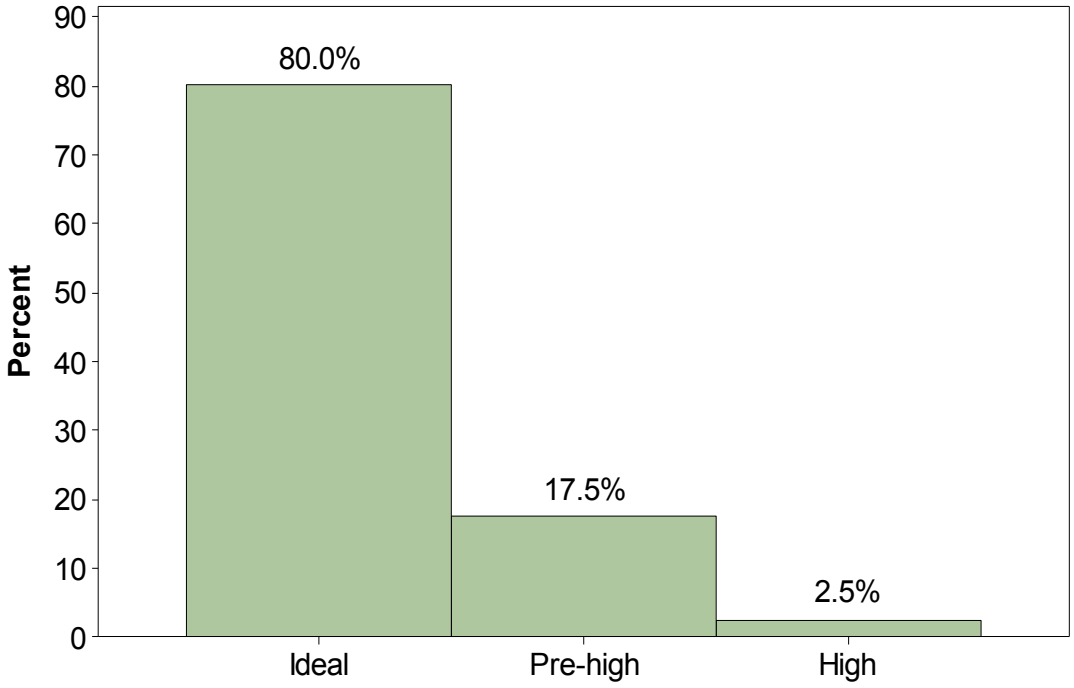

Figure 3.6 Frequency distribution of blood pressure categories

Over three quarter of the participants (n = 32, 80.0%) had ideal blood pressure, but some (n = 7, 17.5%) had pre-high blood pressure. The sample also included one participant (n = 1, 2.5%) who had symptoms of hypertension.

3.12.5 Cholesterol

Table 3.13 presents a summary of the lipoprotein measurements, indicating all the students had optimal levels of LDL-cholesterol (< 100 mg/dL). Their HDL levels were also optimal (> 60 mg/dL) or near optimal (40-59 mg/l). The low relative risk of CVD was indicated by the LDL/HDL ratio, ranging from 1.08 to 1.05. None of the participants had symptoms of hyperlipidemia.

Table 3.13 Summary of lipoprotein (cholesterol) measurements

Lipoprotein	Mean	SD	Min	Max
LDL-Cholesterol (mg/dL)	81.26	3.67	75.80	95.11
HDL (mg/dL)	68.35	4.86	52.62	73.09
LDL/HDL Ratio	1.20	0.11	1.08	1.50

3.12.6 Diabetes

Table 3.14 presents a summary of the blood glucose and insulin levels used to diagnose diabetes.

Table 3.14. Summary of blood glucose and insulin measurements

Measure	Units	Mean	SD	Min	Max
Fasting blood glucose	mmol/L	4.56	0.46	3.30	5.30
Blood glucose two hours after ingesting food	mmol/L	5.00	1.07	3.20	7.80
Fasting plasma insulin	mU/L	7.40	1.26	4.08	9.64

When operating normally the body should restore blood sugar levels to a range of about 4.0 to 6.1 mmol/L. but shortly after a meal the blood glucose level may rise temporarily up to 7.8 mmol/L. The glucose levels of nearly all of the participants fell within this normal range. Two participants (n = 2, 5.0%) however, had blood glucose two hours after meals falling to below 4.0 mmol/L. The fasting plasma insulin levels were within the expected range of around 8.6 mU/ml. All the participants were classified as non-diabetic according to the International Diabetes Federation (2007) target levels for average blood sugar levels.

3.12.7 Consumption of alcohol

Figure 3.7 illustrates the frequency distribution histogram for the consumption of alcohol. The majority of the participants (32, 80.0%) reported that they had not consumed any alcohol in the last three days. Some of the participants (n = 5, 12.5%) had consumed less than the 3 to 4 units of alcohol (24 to 32 g) per day for men and 2

to 3 units (16 to 24 g) per day for women recommended by the NHS. The remaining participants (n = 3, 7,5%) all of whom were male, admitted to exceeding the guideline by drinking an average of 41.6 g alcohol per day, increasing their risk of CVD.

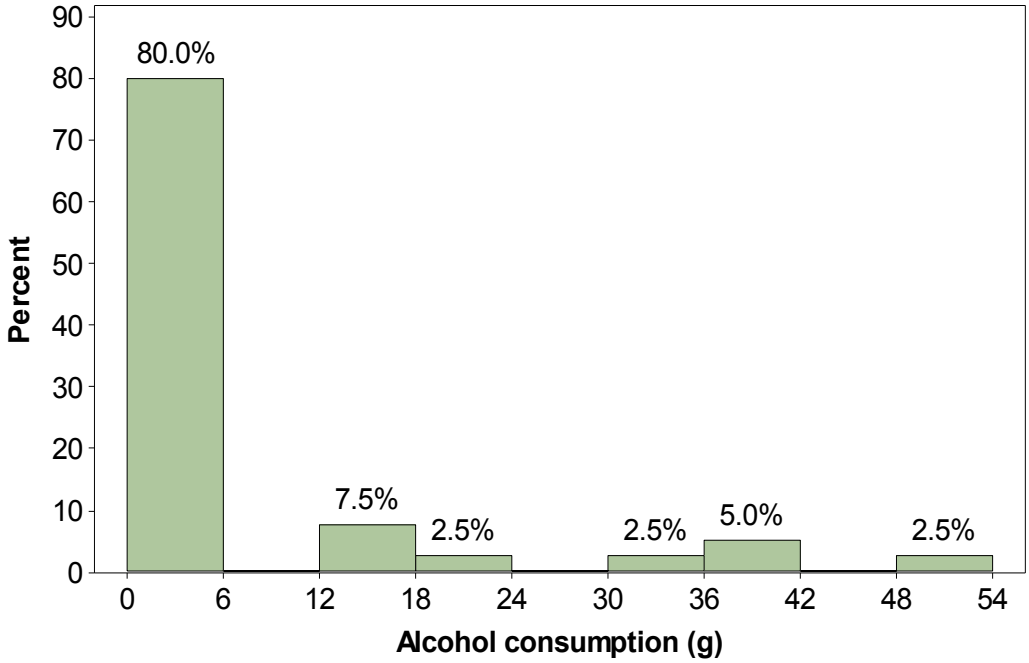

Figure 3.7 Frequency distribution of consumption of alcohol

3.12.7 Smoking

The pie chart in Figure 3.8 illustrates that one tenth (n = 4, 10.0%) of the participants admitted to smoking tobacco, thereby increasing their risk of CVD.

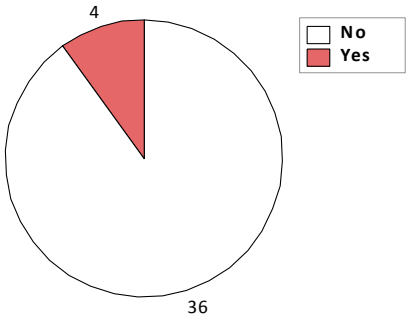

Figure 3.8 Frequency distribution of smoking tobacco

3.13 Multivariate analysis of risk factors

This section presents the results of multivariate statistical analysis using classification, ordination, and partial least squares structural equation modelling (PLS-SEM)

3.13.1 Cluster analysis

Hierarchical agglomerative cluster analysis was conducted, based on a selection of CVD risk factors, including: (a) anthropometric measurements (BMI, body fat, waist to hip ratio); (b) blood measurements (blood pressure, cholesterol, and glucose); and dietary measurements (alcohol, sodium, chloride, saturated fatty acids; non-milk extrinsic sugars, total sugars, and dietary fibre). The cluster analysis converged on a final solution depicted by the dendrogram in Figure 3.9

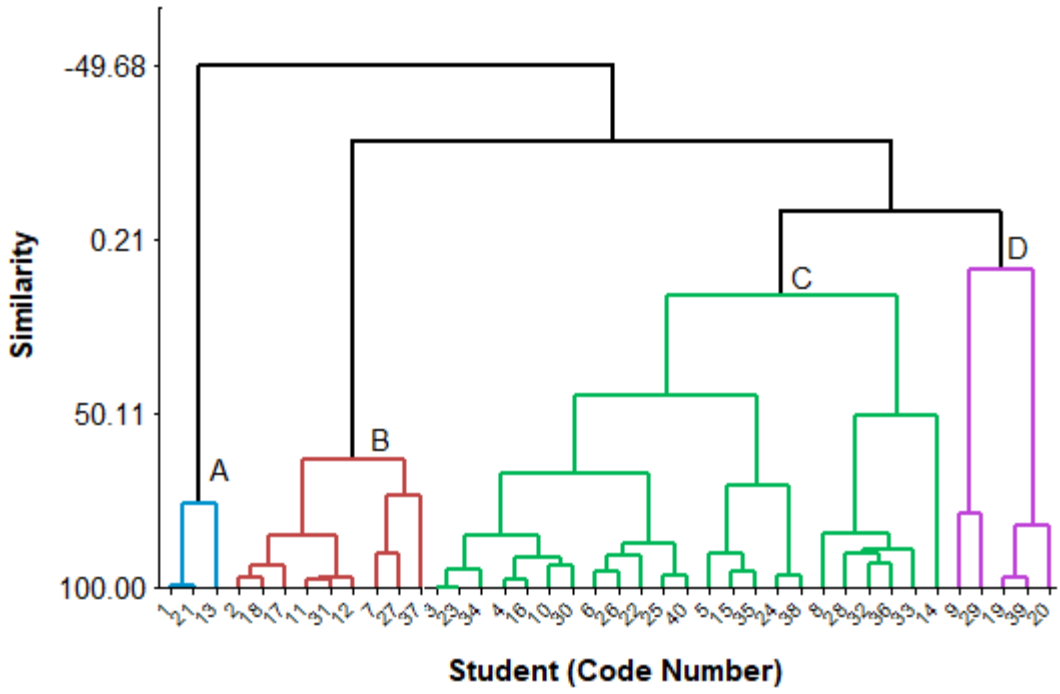

Figure 3.9 Dendrogram to classify 40 students by cluster analysis

The dendrogram identified four mutually exclusive clusters of students, separated by the main (black) branches. The four clusters were designated as Group A (blue), Group B (red), Group C (green), and Group D (purple). Group A and Group D were located at the extreme ends of the dendrogram, meaning the members of these two groups were the most distantly related in terms of their CVD risk factors. The cluster analysis solution provided meaningful results to classify the participants into four separate groups with respect to their CVD risk factors.

3.13.2 Principal component analysis

Figure 3.8 presents the PCA scores computed for the 40 students plotted as points on constructed axes, consisting of three principal components (PC1, PC2, and PC3). The orientation of points in vector space in the 3-D scatter plot indicated the relative similarities and differences between the participants. Closely aggregated groups of points represented closely related individuals, whereas points spaced widely apart, in contrast, represented distantly related or unrelated individuals.

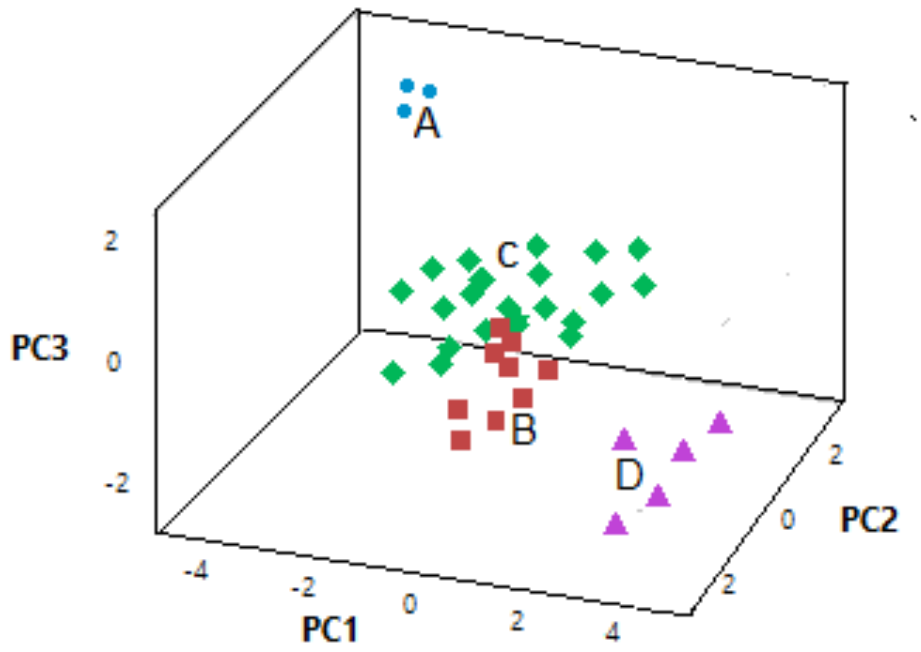

Figure 3.10 3-D scatter plot of principal component scores (PC1, PC2, and PC3). Note: Groups A, B, C, and D are the same as the clusters in Figure 3.7

The orientation of points in the 3-D scatter plot in Figure 3.9 was closely aligned to the four groups of students identified by the cluster analysis in Figure 3.8. The points for group A (blue) were clustered toward the top left-hand side of the plot. The points for group D (purple) were opposed to the points for group A on the bottom right-hand side of the plot. The points for Group C (green) were clustered across the centre of the plot, clearly separated from Groups A and D. The points for group B (red) were clustered at the centre of the plot, sandwiched between groups C and D.

The PCA provided meaningful results to classify the participants into four separate groups with respect to their CVD risk factors. Because the solutions for both cluster analysis and PCA, based on different statistical approaches, were comparable, then the grouping of the participants according to their CVD risk factors were considered to be stable and reliable.

3.13.3 Group profiles

Table 3.15 presents the profiles of the students who were classified into Group A, B, C, or D, based on the mean values of the variables used to conduct the cluster analysis and PCA. Group A was the smallest cluster, with n = 3 members. Group A was characterized by: the highest mean values for BMI (28.10 kg/ m^2); body fat (27.33%); waist to hip ratio (0. 78); systolic blood pressure (128.67 mm Hg); diastolic blood pressure (77.0 mm Hg); LDL-cholesterol (84.00 mg/dL); fasting glucose (4.58 mM/L) and the lowest mean HDL-cholesterol (62.80 mg/dL). Group A also had the highest mean intake of alcohol (9.64 g/d); sodium (3255 mg); chloride (10563 mg); saturated fatty acids (42.83 g); and total sugars (99.04 g); second highest mean intake of non-milk extrinsic sugars (57.90 g); and the lowest mean intake of dietary fibre

(3.31 g). Group A was identified as the cluster with the highest level of CVD risk factors.

Table 3.15 Profiles of CVD risk factors in groups A, B, C, and D (derived by Cluster Analysis and Principal Components Analysis)

CVD risk factor	Group A n = 3	Group B n = 9	Group C n = 23	Group D n = 5
Anthropometric:				
BMI (kg/m^2)	28.10	24.10	23.53	23.49
Body fat (%)	27.33	26.56	19.79	13.01
Waist to hip ratio	0.78	0.76	0.77	0.72
Blood:				
Systolic pressure (mm Hg)	128.67	123.60	121.22	118.33
Diastolic pressure (mm Hg)	77.44	74.80	72.67	69.22
LDL-cholesterol (mg/dL)	84.00	79.10	79.19	79.00
HDL-cholesterol (mg/dL)	62.80	68.68	70.17	70.89
Fasting Glucose (mM/L)	4.58	4.65	4.50	4.53
After meal Glucose (mM/L)	5.23	4.95	5.07	4.66
Diet:				
Alcohol (g)	9.64	5.20	0.78	0.00
Sodium (mg)	3255	2834	2899	2385
Chloride (mg)	10563	3933	4418	3431
Saturated fatty acid (g)	42.83	39.67	32.70	24.43
Non-milk extrinsic sugars (g)	57.90	64.92	54.27	42.40
Total sugars (g)	99.04	69.30	71.00	42.98
Dietary fibre (g)	3.31	6.22	9.34	26.2

Group B consisted of n = 9 members. Compared to Group A, Group B was characterized by lower mean values of BMI (24.10 kg/ m^2); body fat (26.56%); waist to hip ratio (0.76); systolic blood pressure (123.0 mm Hg); diastolic blood pressure (74.80 mm Hg); LDL-cholesterol (79.10 mg/dL); and after meal Glucose (4.95 mM/L); as well as a higher mean HDL-cholesterol (69.68 mg/dL). Compared to Group A, Group B also had a lower mean intake of alcohol (5.20 g/d); sodium (2834 mg); chloride (3933 mg); saturated fatty acids (42.83 g); non-milk extrinsic sugars

(64.92 g); and total sugars (69.30 g) but a higher mean intake of dietary fibre (3.31 g). Group B was identified as the cluster with the second highest level of CVD risk factors.

Group D had n = 5 members. Compared to Groups A, B, and C, Group D was characterized by the lowest mean values for BMI (23.49 kg/ m^2); body fat (13.01%); waist to hip ratio (0.72); systolic blood pressure (118.33 mm Hg); diastolic blood pressure (69.22 mm Hg); LDL (79.00 mg/dL); after meal glucose (4.66 mg/dL) as well as the highest mean HDL (70.89 mg/dL). Group D also had zero intake of alcohol, the lowest intake of sodium (2385 mg); chloride (3431 mg); saturated fatty acids (24.43 g); non-milk extrinsic sugars (42.40 g); and total sugars (42.98 g); but the highest mean intake of dietary fibre (26.2g). Group D had the lowest level of CVD risk factors.

Group C was the largest cluster, with n = 23 members. In Group C, the mean values of the anthropometric, blood, and dietary measures were intermediate between Group A and Group D. Consequently, Group C was identified as a cluster consisting of average students with a moderately low level of CVD risk factors.

3.13.4 Structural equation model

A structural equation model was constructed using SmartPLS. The single endogenous latent variable was “Anthropometric measurements” (a combination of BMI, body fat, and waist to hip ratio). The path coefficients representing the partial correlations between the anthropometric measurements and three exogenous variables were computed for (a) “Unhealthy diet and lifestyle” (a combination of smoking, consumption of alcohol, sodium, chloride, sugars, and saturated fatty acids); (b) “Healthy diet and lifestyle” (a combination of consumption of dietary fibre, fruit and vegetables, and physical activity); and (c) “Blood measurements” (a combination

of blood glucose, blood pressure, and LDL-cholesterol). The path diagram output by the graphic user interface of SmartPLS is presented in Figure 3.11.

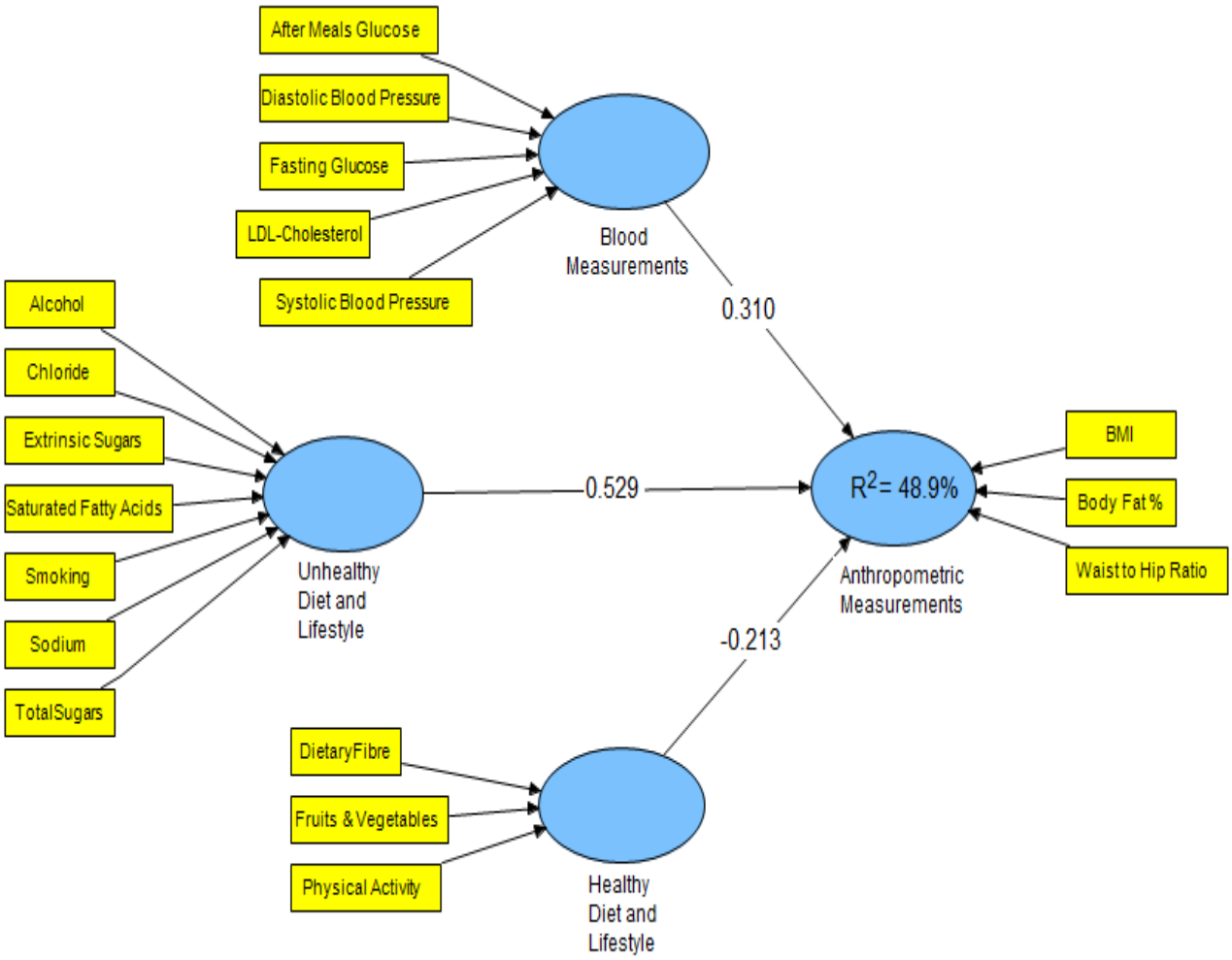

Figure 3.11 PLS-SEM path diagram of relationships between anthropometric and blood measurements, health and lifestyle

The PLS-SEM model explained almost half of the variance (R^2 = 48.9%) in the anthropometric measurements. Using the criteria of Ferguson (2009) this R^2 (between 25% and 64%) represented a "moderate" effect size. The path coefficients indicated that the anthropometric measurements were correlated with both the blood measurements and the diet and lifestyle factors. The unhealthy diet and lifestyle factors were the most strongly correlated with anthropometric measurements, indicated by the largest path coefficient (β = 0.529). The positive coefficient indicated that the anthropometric measurements would increase if the student was a smoker and consumed large amounts of alcohol, saturated fatty acids, salt, and sugars. The blood measurements were less strongly correlated with the anthropometric measurements, indicated by the smaller path coefficient (β = 0.310). This positive coefficient predicted that the anthropometric measurements would be greater if the student had high levels of blood pressure, blood glucose, and LDL-cholesterol.

The healthy diet and lifestyle factors were the least strongly correlated with the anthropometric measurements, indicated by the smallest path coefficient (β = -.213). This negative coefficient predicted that the anthropometric measurements would be smaller if the student consumed high amounts of dietary fibre, consumed at least five servings of fruit and vegetables per day, and also had very active levels of physical activity.

3.12 Discussion

The results of the second survey, based on a convenience sample of 40 students addressed Research Question 1: What are the dietary and lifestyle risk factors for cardiovascular disease among university students in Edinburgh, Scotland. Several CVD risk factors were identified, including obesity, hypertension, unhealthy diet, low physical activity, and smoking tobacco. No symptoms of hyperlipidemia or diabetes were identified.

3.12.1 Obesity

According to the World Health Organisation (2012a), one in ten adults in the world is obese (defined as having a BMI > 30 kg/m^2). The number of overweight and obese children and adolescents has doubled in the last two decades in the Western world (Ogden et al. 2002; 2010). Only 5% of the students sampled in the current study were, however, classified as obese, implying that the study sample may not have been representative. There may be a higher level of obesity in the entire undergraduate population.

3.12.2 Hypertension

According to the British Hypertension Society, the prevalence of hypertension in people over 16 years old in UK is 31.5% in men and 29.0% in women. Only 2.5% of the students sampled in the current study had symptoms of hypertension (i.e., systolic blood pressure ≥ 140 mm Hg and/or diastolic blood pressure ≥ 90 mm Hg.) implying that the study sample may not have been representative. There may be a higher level of hypertension in the entire undergraduate population.

3.12.3 Diet

On average, the participants consumed substantially more saturated fatty acid, sodium, chloride than listed in the dietary reference intakes (DRI) for males and females aged 19-30. Excessive consumption of saturated fats reflects a diet rich in animal sources, including beef, lamb, pork, lard, cream, cheese, and other dairy products. In addition, it may reflect consumption of many baked goods and fried foods. Excessive consumption of saturated fatty acid is known to be a risk factor for CVD in the UK, and therefore its consumption should be reduced (British Dietetic Association, 2012; British Heart Foundation, 2015). The results confirmed the assertion of the National Health Service (2012) that "Most people in the UK eat too much saturated fat".

The average consumption of sodium was in excess of the DRI values. Excessive consumption of sodium reflects a high intake of salt. Many studies have linked dietary salt intake to hypertension, and a reduction in dietary salt intake has been reported to lower blood pressure (Frisoli et al. 2012).

The average consumption of dietary fibre was substantially less than the DRI values. The relatively low consumption of dietary fibre was an important finding, because dietary fibre may have a protective effect on the risk of CVD (Bazzano et al. 2003). The relatively low consumption of polyunsaturated fats was another important finding, because they provide essential fatty acids and fat-soluble vitamins (Crawford, 2013).

The consumption of non-milk extrinsic sugars, found in foods such as sweets, biscuits, cakes, pastries, breakfast cereals, and beverages such as soft drinks, may be a risk factor for CVD (Young et al. 2014). The relatively low consumption of non-milk extrinsic sugars (lower than the DRI values) observed among the students at Heriot Watt University reflected a reduced risk of CVD, and contradicted previous studies suggesting

that the mean intake of extrinsic sugars in UK was significantly higher than the UK recommended population average (Rugg-Gunn et al., 2007; McNeill et al. 2010).

3.12.4 Diabetes and Hyperlipidemia

None of the participants were classified diabetic or pre-diabetic according to the criteria specified by the International Diabetes Federation (2007) target levels for average blood sugar levels. Given that 4.05 million people are diagnosed with diabetes in the UK (Diabetes, UK) the study sample may not have been representative. Furthermore, all of the participants had optimal levels of LDL. Nearly all of the participants (95.0%) had optimal levels of HDL and two had near optimum levels. The LDL/HDL ratio of all of the participants (≤ 1.5) indicated a low risk of CVD. In comparison, as many as two thirds of the UK adult population may have hyperlipidemia (JBS3, 2016).

These results indicated that: (a) the study sample may not have been representative of the UK adult population; and/or (b) the students were too young (18 to 24 years) to have developed Type II diabetes (also known as adult-onset diabetes) and hyperlipidemia, which tend to be age-associated conditions, typically diagnosed in adults over the age of 40 (World Health Organization, 2012b).

3.12.5 Smoking

One tenth (10%) of the participants in the current study admitted to smoking tobacco, thereby increasing their risk of CVD. According to Action on Smoking and Health (2016) the prevalence of smoking in UK in 2013 and 2014 was 19%. Consequently, there was no evidence to confirm that higher levels of cigarette smoking in Scotland may be one of the potential causes of the inequalities reported in CVD prevalence across the UK (Bhatnagar et al. 2015). The students did not display the higher

levels of smoking that may play an important role in the inequalities of CVD between Scotland and other regions of the UK (Popham, 2011).

3.12.6 Analysis of risk factors

Statistical evidence was presented to address Research Question 2: To what extent can the students be classified into groups according to their risk factors for cardiovascular disease. Cluster analysis and principal components analysis separated the participants into four mutually exclusive groups with respect to their CVD risk factors. One group of participants, defined as Group A, was characterized by: the highest mean values for BMI, body fat, waist to hip ratio, blood pressure, LDL-cholesterol, and fasting glucose. Group A also had the highest mean intake of alcohol, sodium, chloride, saturated fatty acids, and total sugars, as well as the second highest intake of non-milk extrinsic sugars. Consequently, Group A, representing 7.5% of the study sample, was identified as the cluster with the highest level of CVD risk factors. In comparison, about 5% of the adult UK population in the UK are estimated to have a high risk of CVD, increasing to 15% among individuals who have elevated systolic blood pressure and cholesterol levels, and over 20% among individuals over the age of 70 (Cardiovascular Risk Assessment, 2012).

Statistical evidence was also provided to address Research Question 3: To what extent are the risk factors related to each other? The CVD risk factors were incorporated into a structural equation model predicting that the anthropometric measurements (a combination of BMI, body fat, and waist to hip ratio) increased with respect to an elevation in unhealthy diet and lifestyle factors (a combination of the consumption of alcohol, sodium, chloride, extrinsic sugars, total sugars, saturated fatty acids, as well as smoking). The anthropometric measurements also increased with respect to an elevation

in the blood measurements (glucose, blood pressure, and LDL-cholesterol). Conversely, the anthropometric measurements decreased with respect to an improvement in healthy diet and lifestyle (a combination of the consumption of dietary fibre and fruit and vegetables, as well as a high level of physical activity). These relationships have already been established in the Framingham model (Wilson, 2013); however, the researcher was the first to identify these relationships using PLS-SEM.

In the tradition of an exploratory research design, the results of the multivariate analysis, including PCA, Cluster Analysis, and PLS-SEM did not attempt prove the existence of causal relationships. The results did not predict that obese people become obese because they have unhealthy diet and lifestyle factors, nor that BMI, body fat, and waist to hip ratio can be axiomatically reduced by adopting a healthy diet and lifestyle. The models were only a basic attempt to summarize the empirical relationships between the anthropometric measurements, dietary, and lifestyle factors using a multivariate approach. The models did not test, confirm, or reject any hypotheses, nor did they indicate the extent to which the observed relationships between the variables were valid and reliable. Unlike the Framingham model (Wilson, 2013); the conceptual model proposed by Arts et al. (2014); and the SEM model constructed by Mi et al. (2011) the models constructed in this study did not attempt to predict the risk of CVD. Like previous models, however, they did focus on the potential importance of bringing together and analysing the relationships between multiple risk factors for CVD (e.g., consumption of alcohol and unhealthy foods, obesity, smoking, blood glucose, cholesterol, and physical activity) as opposed to focusing on only one or two single factors. The construction of this model highlighted the assertion of Young (2009) that simple analyses of data

collected in medical research, involving the analysis only one or two variables are misleading, and that multivariate methods of analysis are essential to identify the relationships between disease related factors.

3.12.7 Limitations

The results of this study were limited by the use of convenience samples. Due to the difficulties involved in drawing random samples from the population, medical researchers frequently employ convenience samples for practical reasons, and often make the assumption that "whatever results are found at the test site, similar results will also be found at other sites and with different subjects" (Kuzma & Bhonenblust, 2005). This assumption is not, however, always justified. It is probable that the 40 students in the convenience sample who volunteered to participated in this study were not representative of the population of undergraduate students at Heriot Watt University. A sample size calculation suggested that to obtain a representative sample from a population of 5796, the sample size, drawn at random from this populations, should be at least 361.

The results of this study are limited because the students who gave their consent to participate consisted of volunteers from one university in Edinburgh, Scotland. Students who volunteer to participate in research may provide different data to those who do not agree to participate, resulting in sampling bias (Fraenkel & Wallen, 2010). Because the sample consisted entirely of volunteers, and was not drawn randomly from the student population as a whole, the findings of this study have limited external validity (i.e., the conclusions may not necessarily be generalizable to all students, at all times, and in all places, but may only be pertinent to the sample of students who volunteered to participate).

One of the key criteria by which medical research is judged is the sample size (Van Voorhis & Morgan, 2007; Zodpey, 2004). The use of sample sizes that are too small is scientifically inappropriate, because the inferential test statistics and p-values computed using classical methods of inferential statistics, such as correlation analysis t-tests, and ANOVA, may be inaccurate and inconsistent (Bachetti et al. 2005; Halparn et al. 2002; Lilford & Stevens 2002; Maxwell & Kelley, 2011). Cohen (1992) recommended that the minimum sample sizes to provide sufficient statistical power to detect a significant correlation at the conventional p = .05 significance level between two variables using Pearson's *r* coefficient is N = 783 if the correlation is small, and N = 85 if the correlation is moderate. Consequently, the sample size of 40 used in the main study provided insufficient statistical power to conduct this type of correlation analysis. Alternative statistical methods had to be used to analyse the relationships between the variables. PLS-SEM was particularly useful, because it did not involve the computation of inferential test statistics and p-values that were a function of the sample size. PLS-SEM was appropriate because it is reputed to be less sensitive to sample size considerations than conventional classical methods of inferential statistics (Hair et al. 2010).

The interpretation of the results of this study was limited by threats to internal validity, defined as the extent to which inferences about cause and effect relationships are influenced by uncontrolled factors (Creswell, 2009). The first threat was that the statistical analysis of variables collected in a survey using modelling techniques do not prove the existence of causal relationships (Pearl, 2009; Reiter, 2000; Bollen & Pearl, 2013). Consequently, the relationships between the blood measurements and

anthropometric measurements using the PLS-SEM model were not causal. It was not possible to determine if cause and effect relationships existed between the identified CVD diet and lifestyle risk factors and the ultimate development of CVD among the students, because the outcomes of the CVD risk factors may not develop for many years.

A further threat to internal validity was the use of self-report questionnaires. The validity of the findings of this study depended on the participants providing accurate self-reported responses to the survey questions. Research has shown, however, that the validity of self-reported measures of dietary intake and physical activity are distorted by social desirability bias (Adams et al. 2005; Bener et al. 2003; Hebert et al. 1995; Scagliusi et al. 2003). Social desirability bias refers to the respondents' desire, at either a conscious or subconscious level, to present a favourable image of themselves, by under-reporting socially undesirable behaviour (e.g., consuming unhealthy food, smoking, drinking alcohol, and having a low level of physical activity) and over-emphasizing socially desirable behaviour (e.g., consuming healthy food, not smoking, not drinking alcohol, and having a high level of physical activity). Socially desirable responding results in in unreliable and inconsistent responses to self-report questionnaires administered to collect data in medical research (Holtgreaves, 2004; Mortel, 2008). The extent to which the findings of this study were distorted by social desirability bias was unknown.

In this study, physical activity was measured by means of a simple self-reported 3-point scale (Less active, Moderately active, or Very active). The validity of this type of ordinal scale for measuring physical activity is relatively poor (Baecke et al. 1982; Ainsworth et al. 1993; Jacobs et al. 1993). Dishman & Steinhardt (1988) found that the

levels of seven-day recall of different types of physical activity among college students were very low. The degree of physical activity is an important lifestyle characteristic, related to the risk of CVD, and it needs to be measured using a more detailed and accurate instrument. There is clearly a need to improve the measurement of physical activity in students (MacKay et al. 2007). Brenner & DeLameter (2014) suggested the use of a chronological reporting procedure using text messaging to improve the accuracy of the measurement of physical activity. An alternative approach could be to provide study participants with accelerometry-based electronic motion detectors that accurately and continuously monitor their physical activity (Yang & Hsu, 2010).

Chapter Four

Conclusions

4.1 Recommendations for practice

The general conclusion of this study is that improvements in the control of risk factors for CVD among University students in Scotland (and elsewhere) need to be made. Several other researchers (e.g. Arts et al. 2014; Biswasit et al. 2015) similarly suggested that there was an urgent need to intervene to bring about a change the unhealthy behaviours of undergraduate students. This recommendation is consistent with the suggestion of Reddy and Kattan (2004, p. 167) that:

> "Sufficient knowledge exists to recommend nutritional interventions, at both population and individual levels, to reduce cardiovascular risk. That knowledge should now be translated into policies which promote healthy diets and discourage unhealthy diets. This requires coordinated action at the level of governments, international organizations, civil society and responsible sections of the food industry".

This conclusion is also consistent with Bhatnagar et al. (2015) who highlighted that prevention measures to reduce risk factors for CVD (specifically unhealthy diet, low physical activity, and high alcohol and tobacco consumption) are still necessary to control regional inequalities and prevent premature mortality. The available evidence provides the rationale and direction to conclude recommendations for practice, and recommendations for further research, as follows. These recommendations are feasible in practice, because randomized control trials have confirmed that structured educational programmes have beneficial effects to change the lifestyles of students, resulting in the prevention of the early development of CVDs (Cheng et al. 2003).

Nurses, clinicians, health promotion experts, and others who are professionally involved in the healthcare of University students should take every opportunity to discuss with students the implementation of preventive lifestyle interventions in order to

maximize their reduction in CVD risk (Matzo, 2008; Mosco et al. 2007). University administrators should also be involved, to emphasize that Universities, like schools, have an obligation to support the students' adoption of healthy lifestyles, and to place health promotion at the heart of the students' activities. This obligation may be guided by the Nutritional Requirements for Food and Drink in Schools (Scotland) Regulations (2008).

4.2 Recommendations for Further Research

The recommendation for further research as a follow-up to the current study is an evidence-based health promotion project. Health promotion is a process of planning, implementing, and evaluating programmes to help individuals and communities acquire and develop the knowledge and skills they require to adopt beneficial health behaviours and to encourage healthy lifestyles (Healey & Zimmerman, 2010). An evidence-based health promotion project should ideally have (a) a specific target population; (b) empirical evidence to support its implementation; (b) specific, measurable goals; (c) a well-defined programme design and time-frame, and (d) a built-in evaluation process to measure programme quality and health outcomes (Inman et al. 2011).

The target population for the proposed health promotion program consists of undergraduate students in Edinburgh, Scotland. The evidence for the implementation of this programme is rooted in the current study, which (a) identified the occurrence of CVD risk factors among this population, and (b) predicted that a high proportion of the variance in the anthropometric measurements (BMI, body fat, and waist/hip ratio of the target population may be the result of lifestyle and diet factors. The goals of the proposed health promotion programme are to produce measurable reductions in the risk factors for CVD among the target population.

The proposed programme design is a Plan-Do-Study-Act (PDSA) or Deming cycle (Deming, 1986) which incorporates a built-in evaluation process. This cyclic design has been recommended as a best practice for the implementation of health promotion programmes (Healey & Zimmerman, 2010). The PDSA or Deming cycle design is widely applied in translational medical research, meaning the conversion of empirical evidence into professional practice in order to ensure that new knowledge and skills reach the individuals and communities for which the research was originally intended (Woolf, 2009).

The proposed programme should initially be conducted at one University in Edinburgh among a defined population of students for a short trial period before it is subsequently developed for implementation across all Universities in Scotland. The proposed PDSA cycle design is outlined in Figure 4.1.

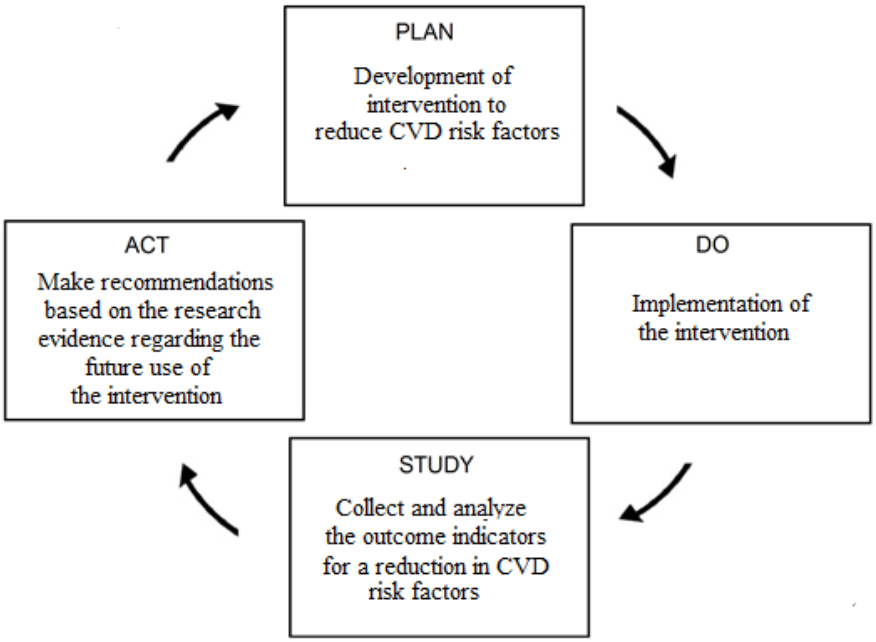

Figure 4.1 Plan-Do-Study- Act cycle for the proposed health promotion project

In the Plan phase, the researcher designs an appropriate intervention, involving a University wide coordinated approach. The intervention team, led by the researcher should consist of University healthcare professionals, as well as a designated health promotion group consisting of academic and academically related staff, who have a vested interest in supporting and developing health awareness within the University. The proposed program should also involve NHS Health Scotland, which is the national health board working with public, private and third sectors to reduce health inequalities and improve health in Scotland. NHS Scotland supports various types of research to inform the direction and development of health promotion activities in Scotland, including surveys, pretesting, and evaluations

The proposed intervention should be underpinned by the Health Belief Model which is a framework for health promotion positing that the perceptions of individuals regarding the severity of, or susceptibility for, unhealthy behaviour is correlated with the likelihood of individuals taking action to reduce the risk of disease associated with that behaviour. Health promotion messages will therefore achieve optimal behaviour changes if they successfully target perceived benefits (Glanz & Bishop, 2010; Jones et al. 2015).

The intervention should also be underpinned by Social Support theory, which posits that changes in human behaviour take place in a social context, and are the consequence of reciprocal relationships between the environment, personal factors, and attributes of the behaviour itself. New patterns of behaviour, including improvements in physical and mental health, are reinforced by the perception that a person has assistance available, or by the actual received assistance, or by the degree to which a person is integrated into a social network (Uchino, 2004).

The intervention team should endeavour to assist each individual student's personal attempts to adopt self-efficacious behavioural patterns by promoting: (a) a weight management program to ensure a BMI of 18.5 to 24.9 kg/m^2; (b) the cessation of smoking and the avoidance of environmental tobacco smoke; (c) moderate physical activity, such as brisk walking every day, to sustain a loss in weight; (d) a diet rich in fruits and vegetables, and high in dietary fibre, and fish at least twice weekly; and (e) the use of dietary supplements, including 850 to 1000 mg of omega-3 fatty acid (Matzo, 2008; Mosco et al. 2007).

Furthermore, the intervention team may consider developing the existing Nutritional Requirements for Food and Drink in Schools (Scotland) Regulations (2008) to provide nutritional guidelines of undergraduates. Table 4.1 summarizes the food standards for school lunches in Scotland. The regulations in Table 4.1 place the sustainable development of health promotion at the heart of a school's activities. Although the food standards are primarily intended for those involved in provisioning food and drink menus in schools, they may also be potentially applicable and adaptable to improve the diet of students eating in University refectories, dining halls, canteens, and at home.

Table 4.1 Food standards for school lunches in Scotland

At a Glance - Food Standards for School Lunches	
1. Fruit and vegetables	A choice of at least two types of vegetables and two types of fruit (not including fruit juice) must be provided every day as part of the school lunch.
2. Oily fish	Oily fish must be provided at least once every three weeks.
3. Variety of extra bread	Additional bread must be provided every day as a meal accompaniment, with a variety of bread, which must include brown or wholemeal, being provided over the week.
4. Oils and spreads	Only oils and spreads high in polyunsaturated and/or monounsaturated fats can be used in food preparation.*
These foods are restricted on your lunch menus	
5. Deep-fried foods	Menus must not contain more than three deep-fried items in a single week (including chips). This includes products which are deep-fried in the manufacturing process. Chips, if served, must be served as part of a meal.
6. Table salt and other condiments	Additional salt cannot be provided. Condiments (if provided) must be dispensed in no more than 10ml portions.
These foods are not allowed on your lunch menus	
7. Confectionery	No confectionery can be provided.
8. Savoury snacks	No savoury snacks can be provided except savoury crackers, oatcakes or breadsticks.

Source: http://www.gov.scot/Publications/2008/09/12090355/4

In the Do phase, the prescribed intervention should be implemented, for a short trial period (e.g., three years, covering the lifespan of one cohort of undergraduate students at the University). In order to reduce risks, and effectively manage available

resources, a PDSA intervention should ideally be implemented on a smaller scale before a longer program can be supported (Langley et al. 2009).

In the Study Phase, the researcher analyses the CVD risk factor indicators collected from each student at the beginning and the end of the programme to determine if significant reductions in CVD risk factors have taken place after the intervention. In the Act phase, the researcher reflects on the findings of the Study Phase, and evaluates the effectiveness of the intervention.

A reduction in CVD risk factors may be identified by analysis of the temporal changes in the measured performances of the students on each of the recommended healthy lifestyle interventions. This analysis will require statistical inferences derived from longitudinal data to determine if there is a significant difference between the pre-intervention data collected in the first year, and the post-intervention data collected in subsequent years. If a significant reduction in CVD risk factors is identified after the intervention, then recommendations can then be made to continue with the intervention. If not, then the next cycle of the PDSA design should be planned, involving the development and evaluation of a potentially more effective intervention. The PDSA cycle may need to continue indefinitely until the target population responds more positively to the intervention in terms of a significant reduction in CVD risk factors.

There are several constraints that may prevent the proposed health promotion program from being implemented, including lack of funds, limited time, and lack of interest from nurses, clinicians, health promotion experts, and others who are professionally involved in the healthcare of University students. The proposed program is feasible if it can be integrated with other health promotion programs currently being

implemented by NHS Health Scotland (the national health board working with public, private and third sectors to reduce health inequalities and improve health in Scotland). For example, the proposed program may be linked to Scottish Government's overarching strategy for tackling obesity, called the "Prevention of Obesity Route Map"

There may be opposition from the students regarding the proposal to change their dietary patterns. Recent research in USA emphasized the problems faced by school policy makers after implementing food standards for school lunches (Government Accountability Office, 2014). After enactment of revised food standards, the authorities providing school lunches faced considerable challenges due to increased costs and increasing amounts of waste. Participation in school lunches declined by one million students between 2010 and 2013. A decline in participation was attributed to the students' negative reactions to the food standards (e.g., they complained that they were provided with a limited variety of food choices, and were given unappealing foods, such as whole grains, that they did not like to eat, resulting in large amounts of waste). The students missed the options to eat pizza, sandwiches, potato chips, and deserts at lunch. Other complaints included the poor quality and taste of the food, and the limited availability of flavour enhancers (e.g., salt and condiments).

4.4 Final conclusion

The conclusions of the current study, including the recommendations for further research, assume that the collection and analysis of quantitative data can provide definitive answers to research questions concerning (a) the identification of CVD risk factors; and (b) the extent to which these risk factors are inter-related and can be alleviated. This assumption, however, is questionable, because quantitative analysis aims

to generate conclusions that can be generalized from the sample to a population norm (e.g., using mean values); however, statistics cannot explain why so many individuals in a population may behave differently from the norm (Banerjee & Chaudry, 2010). Every individual in a population is genetically unique, and may respond differently to CVD risk factors, because some genes they carry may potentially protect them from CVD (Mi et al. 2011). Genomes, polymorphisms and alleles associated with cholesterol metabolism which are unique to each individual may change the risk of CVD (Kathiresan et al. 2008; Whitfield, 2014). The grouping of patients according to the mean values of their risk factors is therefore subject to error associated with the ecological fallacy. The ecological fallacy, which is a serious issue in medical and healthcare research, implies that statistics computed to describe a group do not necessarily apply to every member of that group (Diez- Roux, 1998; Idrovo, 2011). Deriving conclusions about how to reduce the CVD risk factors for every individual student in a population, based on multivariate statistics that applied to a sample of students classified into groups, is therefore very difficult.

Consequently, it is recommended that future research on CVD risk factors should use a mixed methods approach, implying a combination of both quantitative and qualitative methodologies. Although sometimes dismissed as a less scientific approach, qualitative methodologies are particularly valuable in medical and healthcare research because they facilitate the collection of rich and accurate details of the lived experiences of individuals, in order to reveal the intrinsic beliefs, values, and motivations associated with the health behaviours of each individual (Al Busaidi, 2008; Pope & Mays, 2006; Rahman & Majumder, 2013). For example, by interviewing a group of students and conducting a content analysis of the interview transcripts, qualitative themes could be

extracted that might explain why certain students choose to adopt healthy lifestyles and consume healthy food, whereas others choose not to do so (Tonon, 2015).

A combination of quantitative and qualitative research methodologies has not previously been applied to examine CVD risk factors and how they can potentially be alleviated. It remains to be seen whether a mixed methods approach might help to explain the variability of CVD risk factors among University students, and ultimately provide evidence to support the development of an effective health promotion programme that will help to alleviate CVD risk factors among a larger proportion of University students in the future.

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Appendix A

Diet and Health Screening Questionnaire

What is your sex?
How old are you?
What is your weight (kg)?
What is your height? (m)
Would consider your self to be ?
How do you rate your emotional health?
How do you rate your physical health?
Do you consider your self to be? [Very active person; Moderately active person; Less active person)
Health: Is there a family history of one of the following diseases? [Diabetes?]
Health: Is there a family history of one of the following diseases? [High blood pressure or hypertension?]
Health: Is there a family history of one of the following diseases? [High cholesterol level?]
Health: Is there a family history of one of the following diseases? [Heart diseases, stroke?]
Diet: [Are you vegetarian?]
Diet: [Are you vegan?]
Diet: [Are you lactose intolerant?]
Diet: [Do you have breakfast everyday?]
Diet: [Do you eat 5 servings of fruits/vegetables everyday?]
Diet: [Do you take any dietary supplements (such as Vitamins and Minerals?]
Life style: [Do you smoke?]
Life style: [Do you walk for at least 10 minutes everyday?]
Life style: [Do you do strong physical activities 3 times a week such as aerobics, running, fast bicycling or fast swimming?]
Life style: [Do you do moderate physical activities 3 times a week such as bicycling at a regular pace, swimming at regular pace?]
Beverages: [Do you drink soft drinks?]
Beverages: [Fruit juice or other juices]
Beverages: [Sweetened coffee or tea drinks like a Frappuccino, Frappe, or Chai (do not include unsweetened coffee or tea]
Beverages: [Unflavoured milk]
Beverages: [Flavoured milk]
Beverages: [Alcohol drinks]
Beverages: [Sport drinks (such as PowerAde, Lucozade)]
Beverages: [Energy drinks (such as Red Bull); these drinks usually have caffeine]
Beverages: [Diet soft drinks (include all kinds such as Diet Pepsi, Diet Irn Bru, Diet Coke, Diet 7-Up)]
Beverages: [Regular soft drinks (include all kinds such as Coke, Pepsi, 7-Up, Sprite, Irn Bru)]
Beverages: [330ml (normal can)]
Beverages: [500ml (large individual bottle)]
Beverages: [2 Lt (larger family bottle)]